A CLINICAL STUDY, DIAGNOSIS AND MANAGEMENT OF LIVER ABSCESS

DISSERTATION SUBMITTED FOR

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY, CHENNAI

With partial fulfillment of the regulations for the award of the degree of

M.S (General Surgery)

Branch - I

Government Kilpauk Medical College

Chennai – 600 010.

April -2017

CERTIFICATE

This is to certify that this dissertation titled **"A CLINICAL STUDY ,DIAGNOSIS AND MANAGEMENT OF LIVER ABSCESS"** submitted by **DR.JAGANMURUGAN R** to the TamilNadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of MS degree Branch I General Surgery, is a bonafide research work carried out by him under our direct supervision and guidance from january2016 to september 2016.

Prof. Dr. R.KANNAN, M.S.,

Professor & Head of the Department,
Department of General Surgery,
kilpauk Medical College,

chennai.

Prof. Dr. R.KANNAN, M.S.,

Professor & Unit Chief
Department of General Surgery,
kilpauk Medical College,

chennai.

Prof.R.NARAYANA BABU, M.D, DCH.
DEAN
Govt. Kilpauk Medical College,
Chennai-10

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation titled **"A CLINICAL STUDY ,DIAGNOSIS AND MANAGEMENT OF LIVER ABSCESS"** is a bonafide research work done by **Dr.JAGANMURUGAN R**, post graduate in M.S. General Surgery, Kilpauk Medical College, Chennai-10 under my direct guidance and supervision in my satisfaction, in partial fulfillment of the requirements for the degree of **M.S. General Surgery**.

Date:

Place:

Prof.R.KANNAN, M.S.,
Professor of General Surgery,
Kilpauk Medical College,
Chennai-10

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Date:

Place:

Prof.R.KANNAN, M.S.,
Professor of General Surgery,
Kilpauk Medical College,
Chennai-10

DECLARATION

I**, DR.JAGANMURUGAN R** solemnly declare that,I carried out this work on **"A CLINICAL STUDY ,DIAGNOSIS AND MANAGEMENT OF LIVER ABSCESS"** at the Department of general surgery,Govt royapettah hospital Kilpauk medical college during period of 2014 to 2016 . I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award,degree,diploma to any other university ,board either india or abroad. This is submitted to **The TamilNadu Dr. M.G.R. Medical University, Chennai,** in partial fulfillment of the regulations for the award of MS degree (Branch I) General Surgery.

Place: Chennai **DR.JAGANMURUGAN R**

Date:

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I would like to thank God for the things he has bestowed upon me.

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INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10

Protocol ID. No. 6/2016 Dt: 23.01.2016

CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A clinical study diagnosis and management of liver abscess" - For Project Work submitted by Dr.R.Jaganmurugan, PG Student of MS (General Surgery), Govt. Kilpauk Medical College, Chennai-10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

DEAN
Govt.Kilpauk Medical College,
Chennai – 10.

INTRODUCTION

Hippocrates described about liver abscess in 460-377 B.C. , still it remains a challenging situation because of its highly variable presentation, leading to diagnostic difficulties.

Tropical country like india has 400 million people harbouring E.histolytica that causes amoebic liver abscess , it requires immense importance for thorough understanding of the same.

Among the developing countries worldwide, India has 2nd highest incidence of liver abscess . Liver abscess is term for collection of purulent material in liver parenchyma which is due to bacterial , fungal ,parasitic or mied infection. Among all, pyogenic abscesses accounts for four fifth of liver abscess in developed countries, whereas amoebic liver abscess account for two third of liver abscess in developing countries.

Amoebiasis is presently the third most common cause of death from parasitic disease . The condition is endemic in India because of overcrowding and poor sanitary condition .3–9% of all cases of amoebiasis produce liver abscess. However,other etiologieslike pyogenic and tubercular should always

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colonic amebiasis as the antecedent source of liver abscess,provided the basis for management of amoebic liver abscess. Early treatment with open surgical drainage alone had limited success rate . Efforts to treat both liver abscess and colonic infestation improved the success rate. systemic amoebicidal agents along with USG guided closed aspiration is the treatment of choice. The present laparoscopic era has reduced the open procedure .

Surgical management was the mainstay for treating LA earlier [1]. However, recent evidences from percutaneous drainageprocedure have showna favourable outcome with less average length of stay in hospital compared to conservative mode of treatment [4]. In this context, precise diagnosis of the abscess aetiology is pivotal for appropriate management. The concept of the present study was to evaluate the changing trends in clinical profile, microbiological aetiology, and management outcomes of patients diagnosed with LA

AIMS AND OBJECTIVES

1. To study the Demographic profile

2. To study the risk factors associated with liver abscess .

3. To study the microbiological diversity in liver abscess .

4. To study the spectrum of clinical presentation

5. To evaluate efficacy of Ultrasonographic studies in determining the etiology which may change the treatment outcome

6. To study the effectiveness of different modes of management.

REVIEW OF LITERATURE

Historical aspects

The liver is the organ most subjected to the development of abscesses. In a study about intraabdominal abscesses over a 12 year period liver abscesses made 48% of all visceral abscesses of 540 cases.

Liver abscess was first drained in the Hippocratic era, and master of medicine successfully practiced the draining of pus. The history of amoebiasis goes back to the era of Susruta who gave description of Athisara.as amoebic dysentery .

Lambi described the parasite first and koch demonstrated in pus from the tissues adjoining the liver abscess . Councilman and Lafleur (1891) in Baltimore proved the clinical and pathological evidence that amoeba was responsible for liver abscess.

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In 1918 Roger in his famous paper described “The protozoal organism reaches the liver by portal circulation and they entangle in the interlobular veins producing congestion of liver, he established that amoebae are constantly present in the walls of the abscess though not frequently in pus.”

Oschner & Debakey described Pyogenic liver abscess in 47 cases . In their classic paper and reviewed the world literature in 1948 .

M’Fadzean with his associates in 1953 advocated closed aspiration .He also used antibiotics for treatment of solitary pyogenic liver abscess .

Pyogenic abscess was initially described by Waller in 1846 as a diseased characterized by suppurative thrombophlebitis of the portal vein and formation of single or multiple abscesses

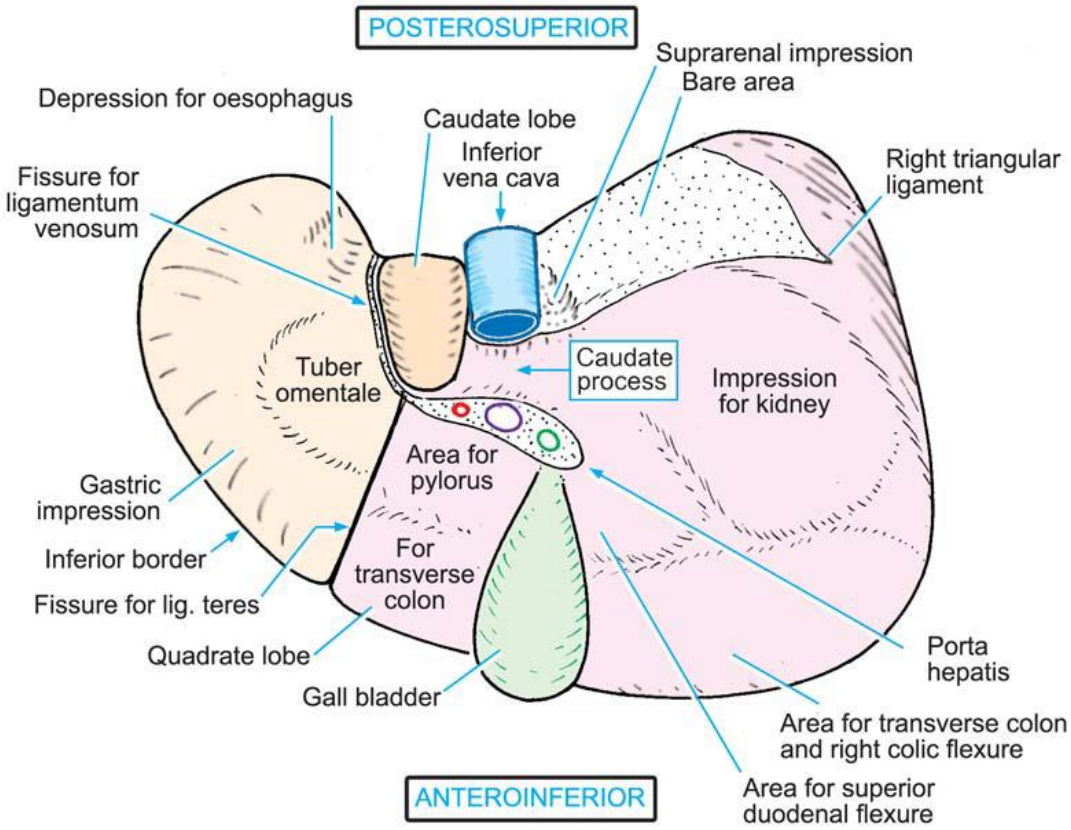
ANATOMY

The liver, the largest gland in the body weighs approximately 1500g and receives about 25% of cardiac output. This wedge shaped organ occupies most of the right hypochondrium and epigastrium. It has visceral and diaphragmatic surfaces. The diaphragmatic surface, convex is divided into anterior, posterior and right surfaces. Sharp inferior border separates right and anterior surface from visceral surface.

The visceral surface contains porta hepatis where the major vessels and duct enter and leave it , but hepatic vein emerges from the diaphragmatic surface.

Ligamentum teres notches the inferior border. The falciform ligament ascends on the anterior surface to reach the superior surface where a reduplication of the left leaf forms the left triangular ligament. The upper layer of the coronary ligament is the right leaf.

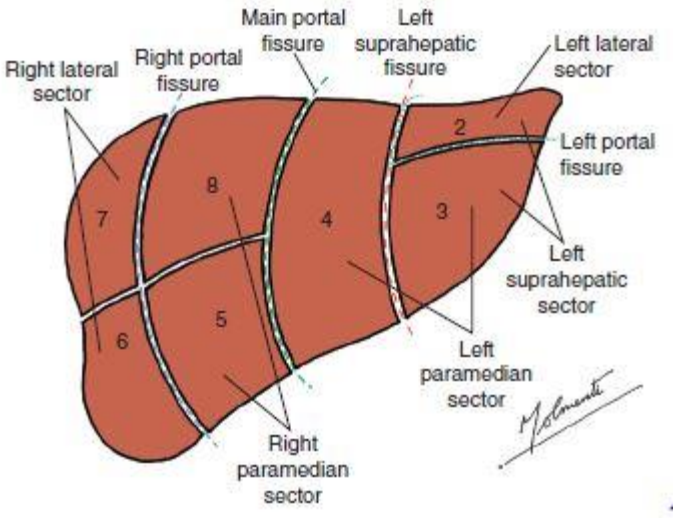
The posterior surface has deep groove in which lies inferior venacava . To the right is the triangular bare area, with the vena cava at its base and with sides formed by superior and inferior layers of coronary ligament. These two layers meet at apex is the right triangular ligament.



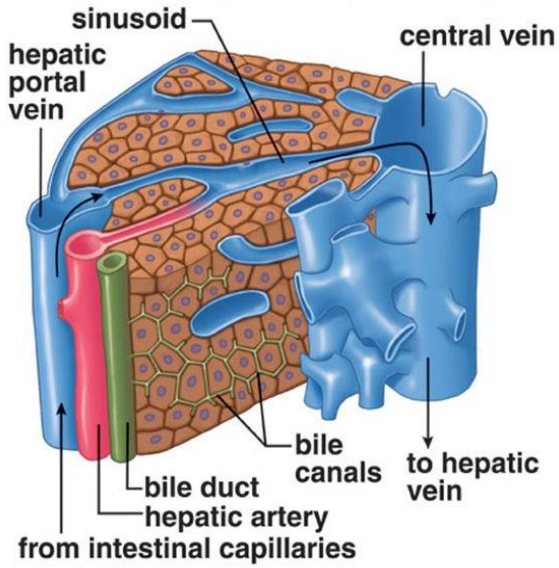
Couinaud's functional segments of Liver

At the porta hepatis, lie the hepatic ducts,hepatic artery and portal vein . From anterior to posterior lies vein-artery-duct. There are also nodes and nerves of the liver. The bare area of liver is in contact with the right suprarenal gland and diaphragm. The surface of liver which is related to stomach, duodenum, hepatic flexure of colon and right kidney is the visceral surface.

SEGMENTS OF LIVER :On the basis of blood supply and biliary drainage there are four main hepatic sectors: left lateral, left medial, right anterior and right posterior



HISTOLOGY



12-33

It is three dimensional lattice composed of parenchymal cells arranged in anastomosing and branching plates.

Portal triad or portal areas or portal canal contains a branch of portal vein, a branch of hepatic artery and an interlobular bile ductule.

In humans, liver contains 3-6 portal canals per lobule. Between parenchymal plates are sinusoidal blood spaces.

Sinusoids are irregularly disposed, normally in a direction perpendicular to the lines connecting central veins.

Walls of the sinusoids consist of endothelial cells called Kupffer cells. Potential spaces between hepatic cells and walls of sinusoids are called space of Disse. .

This space is continuous with larger space that surrounds the portal areas known as the space of Moll.

PYOGENIC LIVER ABSCESS:

Incidence:

Pyogenic liver abscess (PLA) may be defined a" solitary or multiple collections of pus within the liver due to bacterial infection".

In 1938, Ochsner reported the first series of patients with hepatic abscesses in the modern surgical era treated by surgical drainage. The minimally invasive treatment of liver abscess first landmark.It was done by M'Fadzean in 1953.

The development of clinical ultrasound in the 1960s.

The introduction of computed tomography in the 1970s . The two major advances in the diagnosis and treatment of PLAs. Currently, percutaneous needle aspiration and percutaneous catheter drainage have become standard methods for both single and multiple PLAs.

In studies, the most common cause was found to be cryptogenic.That means with no obvious predisposing cause was identified.The incidence of primary cryptogenic PLA is on increase.

Etiology :

Mostly due to infection in biliary or intestinal tracts. Causes of liver abscesses have been divided into six categories .

Hepatobiliary
Cholelithiasis
Benign strictures
Acute cholangitis
Periampullary tumors
Gallbladder cancer

Portal
Diverticulitis
Anorectal suppuration
Pelvic suppuration
Postoperative sepsis
Intestinal perforation
Pancreatic abscess
Appendicitis
Chronic inflammatory bowel disease
Colonic cancer
Gastric cancer

Arterial
Endocarditis
Vascular sepsis
Ear, throat, nose, or dental infection

Traumatic
Open or closed abdominal trauma
Chemoembolization
Percutaneous ethanol injection or radiofrequency ablation

Adjacent Abdominal Pathology
Acute cholecystitis
Gastroduodenal perforation
Colonic perforation

Cryptogenic

Author, Year	No. of Cases	Cryptogenic, %	Hepatobiliary (%)	Portal (%)	Hepatic Artery (%)	Other (%
Oschner et al, 1938[2]	47	60	6	19	N/A	15
Pitt and Zuidema, 1975[8]	80	20	51	15	1	<10†
Branum et al, 1990[5]	73	27	31.4	18.2	10	14‡
Huang et al, 1996[10]	153	16	60	<10	10	<10†
Seeto and Rockey*, 1996[9]	142	40	37	11	N/A	12*
Alvarez et al, 2001[11]	133	26	25§	13	2	33¶
Mohsen et al, 2002[12]	65	24 (18 uninvestigated)	28	48	N/A	N/A
Wong et al, 2002[13]	80	N/A	61	N/A	1.25	N/A

Pathology:

Portal, traumatic, and cryptogenic liver abscesses are solitary and large . Biliary and arterial abscesses are multiple and small. If the primary lesion is located within the portal circulation, usually the abscesses are large, single or multiple and in most cases confined to the right lobe of liver.The left lobe is rarely affected.

In a study based on experiments by Kenny of serege in 1901. The right lobe of liver receives a separate flow of blood from superior mesenteric vein. a The left lobe of the liver from splenic vein. This explains right lobe is preferred location of portal liver abscesses .If the portal vein is with a septic thrombus leads to liver abscess of both lobes.

Fungal abscesses characteristic are multiple, miliary andbilateral .

Microbiology:

On literarture evidence, only 50% positivity in both abscess culture and blood culture was found. This probably due to the result of poor culture techniques. Abscess due to biliary or gastrointestinal source will be polymicrobial. Most common pathogen for cryptogenic abscess in Asians is Klebsiella .Most common anaerobes is bacteroides

Most tuberculosis lesions of the liver are miliary granulomas. Sometimes tuberculomas are formed and spread along the walls of the intrahepatic bile ducts .

Table 2. Microbiologic etiologies of pyogenic liver abscess*

	%
Gram-negative enterics	
E. coli	20.5
K. pneumonia	16.0
Pseudomonas sp.	6.1
Proteus sp.	1.3
Others	7.4
Gram-positive aerobes	
S. milleri	12.2
Enterococcus sp.	9.3
S. aureus / S. epidermidis	7.7
Streptococci sp.	1.1
Anaerobic organisms	
Bacteroides sp.	11.2
Anaerobic / Microaerophilic Streptococci	6.1
Fusobacterium	4.2
Other anaerobes	1.9
Miscellaneous	
Actinomyces	0.3
C. albicans	0.3

Disseminated granuloma inguinale produce miliary abscess.Clostridial infections cause gas abscesses, but most of the jaundice in disseminated infections is hemolytic.

Clinical Features

Most patients with pyogenic liver abscesses present with symptoms of less than 2 weeks duration. The most common presenting symptom is fever. Pain is the next common symptom. Chills and weight loss occur in 50%. Other symptoms like jaundice, diarrhoea, cough, anorexia can also be present.

The most common physical sign is an enlarged tender liver.

	% of Pyogenic Abscesses
Symptom	
Fever	83
Weight loss	60
Pain	55
Nausea and vomiting	50
Malaise	50
Chills	37
Anorexia	34
Cough or pleurisy	30
Pruritus	17
Diarrhea	12
Sign	
Right upper quadrant tenderness	52
Hepatomegaly	40
Jaundice	31
Right upper quadrant mass	25
Ascites	25
Pleural effusion or rub	20

LAB INVESTIGATIONS :

The most common LFT abnormality in liver abscess is elevated alkaline phosphatase. This is seen in 80 -90% of patients. Bilirubin is increased in 40-60% of patients. Transaminases are also elevated. Hypoalbuminemia is seen in 70% of patients . Mild elevation of prothrombin time is also seen.

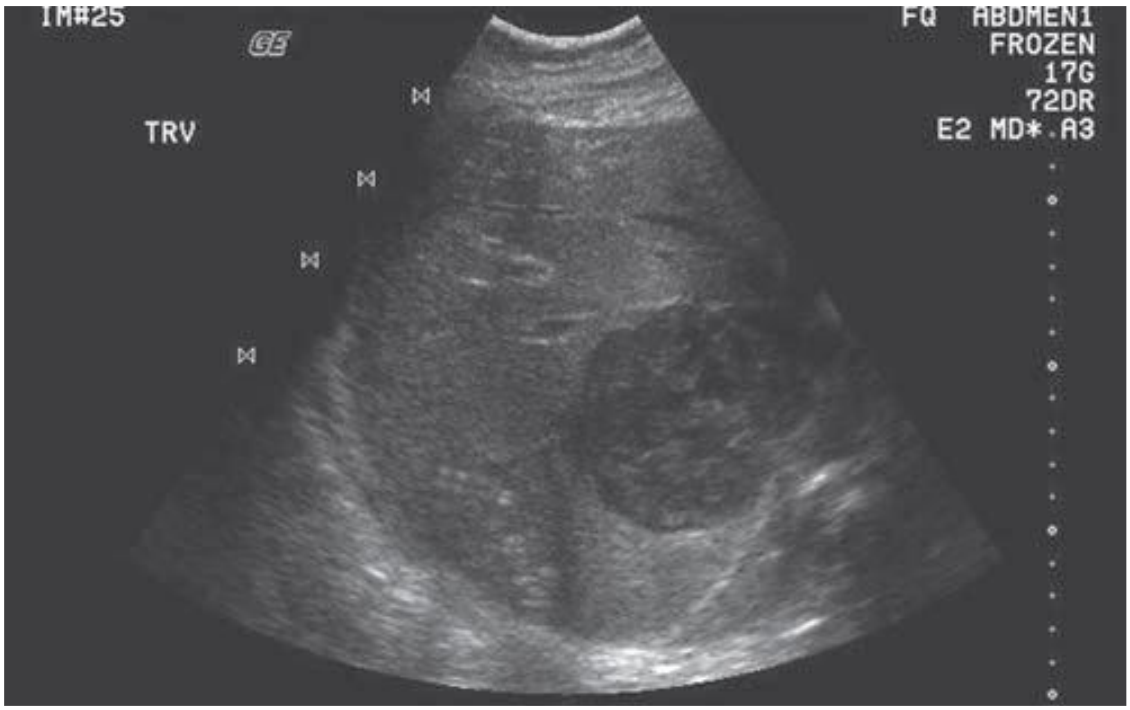
Laboratory data	
Increased alkaline phosphatase	87
WBC count >10,000/mm^3	71
Albumin <3 g/dL	55
Hematocrit <36%	53
Bilirubin >2 mg/dL	24

Radiological investigations:

Chest X ray are abnormal in nearly 50% of patients with liver abscesses. Changes include elevated hemi diaphragm; pleural effusion and lower lobe atelectasis. Gas-forming organisms are present then air fluid level may be seen. unoperated biliary tree with air may also be present.This confirms the diagnosis of cholangitis. portal venous gas seen on an abdominal x-ray, confirms pyelophlebitis.

Portal venous gas - branching linear lucencies in the peripheral portion of the liver .In biliary causes of liver abscess diagnosis requires cholangiography . Either ERCP or MRCP were helpful . It defines biliary anatomy and outline the abscess cavities in about 2/3 of the studies.

USG is the first investigation of choice for diagnosis. USG is 80-95% sensitive. Technetium-99 sulfur colloid scan will show the defect in over 80% of all cases. Other scanning with indium Ill labelled leucocytes and gallium 67 are used.



The pitfalls of USG are:

1. Fatty infiltration causes markedly echogenic liver, makes small abscess undetectable.

2. Multiple microscopic abscesses due to cholangitis not seen separately

3. Cannot visualize the liver dome and may miss lesions there

Diagnostic aspiration or therapeutic drainage can be done by ultrasonography.Computed tomography will distinguish hepatic collections as small as 0.5cm . Multiple small abscesses are seen in CT. Intravenous administration of contrast material enhances the case by which abscesses can be diagnosed. MRI has recently been used for the detection of hepatic abscesses.

TREATMENT

After confirming diagnosis of liver abscess ,then suspected broad spectrum intravenous antibiotics should be started. Antibiotics therapy are adjusted according to cultures . Blood culture is sent. Specimens cultured for acid fast bacilli and fungi . It is done in clinical suspicion of mycobacterium , fungal infections or immune suppressed. Empirical antibiotic therapy should cover against aerobic gram-negative bacteria.

Antibiotics therapy - aminoglycoside or clindamycin with ampicillin or vancomycin. Fluoroquinolones is given instead of aminoglycoside . Metronidazole given for clindamycin, if amoebiasis is suspected. Single-agent therapy with ticarcillin-clavulanate, imipenem-cilastatin, or piperacillin-tazobactam is given. Duration of therapy is 4–6 weeks.

Since the antibiotic penetration into the abscess cavity is often poor and 2 weeks of intravenous antibiotics are usually recommended. Appropriate oral antibiotics are usually continued for a further 4 weeks.

ASPIRATION AND PERCUTANEOUS DRAINAGE:

The Bertel in 1986 published a series 39 patients with pyogenic hepatic abscess. 23 patients were treated surgically .16 patients underwent percutaneous drainage. 3 of the percutaneously treated group required surgical drainage due to viscous abscess content. Mortality was 17% in the surgical group . 13% mortality in percutaneously drained group . Contraindications to catheter drainage are ascites, coagulopathy and close to vital structures.

Table. Comparison between Percutaneous drainage (PD) and Surgical drainage (SD) (reported series).

Authors	Procedure	No. of patients	No. of abscesses	Success Rate (%)	Complications Rate (%)	Mortality (%)
Johnson et al[14]	PD	27	27	89.0	4.0	11.0
(1981)	SD	43	43	79.0	16.0	26.0
Glass and Cohn[15]	PD	15	15	47.0	6.0	Not mentioned
(1984)	SD	44	44	88.0	27.0	Not mentioned
Olak et al[16]	PD	24	24	70.3	30.8	11.0
(1986)	SD	24	24	85.3	15.9	7.4

RCT by Rajak in 1998 for aspiration vs catheter drainage was done. It showed percutaneous aspiration is preferred to catheter drainage. Advantages of aspiration is less invasive and less expensive .Also aspiration avoids the problems related to follow-up catheter care or loss of catheter position. Disadvantages are incomplete evacuation of the abscess cavity and rapid reaccumulation of abscess. Percutaneous aspiration appeared to be less effective than PCD.

Percutaneous drainage is inappropriate in

(1) multiple large abscesses;

(2) known intra-abdominal source that requires surgery;

(3) unknown etiology;

(4) ascites;

(5) abscesses that require transpleural drainage

SURGICAL DRAINAGE:

Ochsner in 1938, published tat surgical drainage reduces mortality. Extra peritoneal drainage is recommended . So as to avoid contamination of the peritoneal cavity. This was achieved by posterior approach of the undersurface of the 12 rib.

The advantages of transperitoneal approach include :

(1) treat the inciting pathology in the remainder of the abdomen/pelvis;

(2) gain access and exposure of the entire liver ;

(3) access the biliary tree for cholangiography and bile duct exploration.

Surgical drainage done for

1) failed nonoperative therapy,

2) surgical treatment of the underlying source,

3) multiple macroscopic abscesses,

4) steroids, or

5) concomitant ascites

Laparoscopic drainage is an attractive alternative for patients requiring open surgical drainage. The advantages of laparoscopic surgery in terms of reduced analgesia requirements, reduced morbidity, faster postoperative recovery and shorter hospital stay compared to laparotomy are well documented. Laparoscopic US is also likely to be useful in this respect.

Liver resection may be indicated in patients with

1)hepatholithiasis, 2)complex bile strictures, or3) liver atrophy.

Outcome:

Surgical drainage and systemic antibiotics decreased the mortality . In the 1980s, availability of US and CT facilitated earlier diagnosis.It also lead to the development of percutaneous methods of drainage. This resulted in a further fall in mortality. Johns Hopkins series, the overall mortality in the period 1952 to 1972 was 65%. Compared to a mortality of 31% during the period 1972 to 1993. Branum in 1990 have reported a mortality of 19% between 1970 and 1986 · Seeto and Rockey in 1996 have reported a mortality of 11% for patients presenting between 1979 and 1994 .

FACTORS ASSOCIATED WITH POOR OUTCOME

Age >70 y	WBC count >20.000/mm^3
Diabetes mellitus	Increasing bilirubin
Associated malignancy	Increasing SGOT
Biliary etiology	Albumin <2 g/dL
Multiple abscesses	Aerobic abscess
Septicemia	Significant complication
Polymicrobial bacteremia	

AMOEBIC LIVER ABSCESS

EPIDEMIOLOGY

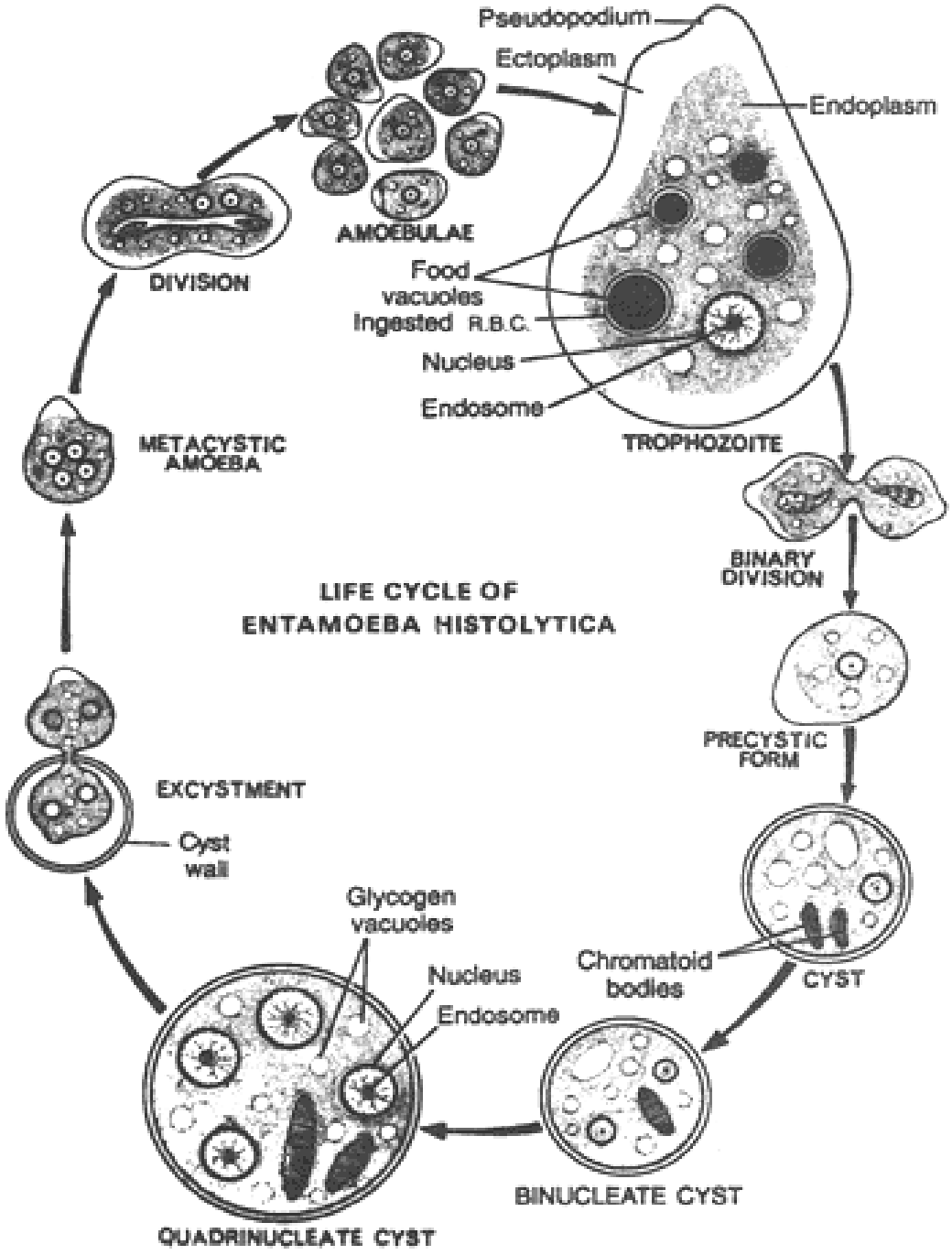
E. histolytica affects 1/10 of the world's population . It is responsible for at least 100,000 deaths per year . Most infections occurring in the developing countries of the tropics and subtropics. 500 million people are carriers of *E. histolytica* or *E. dispar* . 50 million people have active disease.

Infection prevalence varies greatly and in some regions exceeds 50%. One study from Gambia, West Africa documented infection rates approaching 100% annually. Amoebiasis follows a bimodal age distribution.

The new organisms among the entamoebae species are E. Moshkovskii and E.Dispar .They have been described in Indian population.

The association between amebiasis and warm climates results from the poor sanitation and lack of hygiene that accompany poor living conditions. Infection occurs mainly by the fecal- oral route. Contaminated food, unhygienic handling of food and raw sewage contamination water supplies occasionally causes infection.Male homosexuals transmit the infection but harbour nonpathogenic *E. dispar*. *E. histolytica* transmitted by heterosexual activity and also homosexual activity

LIFE CYCLE OF E.*HISTOLYTICA*



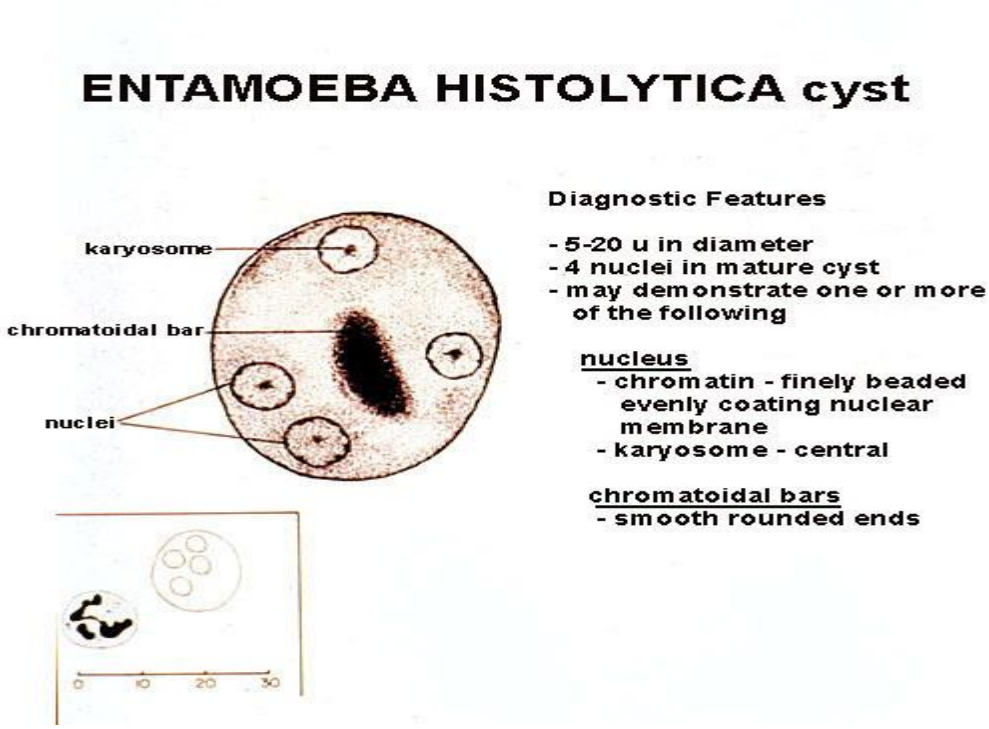
Entamoeba histolytica : Reproductive and life history

Microbiology:

E. histolytica belongs to Sarcodina (has pseudopodia) and the order Amoebida. The genus Entamoeba includes the species E. histolytica, E. bartmanni (a non-invasive 'small race'), E coil, E polecki (pigs) and E. moshkovski (a free-living non-pathogenic). The species are regarded as non-pathogenic, except for E. histolytica.

Protozoan E. Histolytica has two forms: 1)Trophozoite and 2) cyst. The Trophozoites are facultative anaerobes. It has double-layered limiting membrane and 20-30 nn glycocalyx. E. histiolytica are capable of tissue invasion and contact lysis of cells.

Electrophoretic patterns of amoebic enzymes such as 1)glucose-phosphateisomerase,2)I-malate,3)NADPoxidoreductase, 4)phosphoglucomutase and 5)hexokinase.18 zymodenes of E. histolytica have been described from various areas of the world. Seven of these strains have been isolated from subjects with mucosal ulceration and liver abscess and are consequently labeled as pathogenic



It can survive up to 45 minutes in fecal material in nails.Also 1 month in soil at 10°C. They remain infective in fresh water, sea water and sewage .They are destroyed by drying, 200 p.p.m. of iodine and heat > 68°C. cyst are resistant to chlorination used to purify ordinary drinking water.

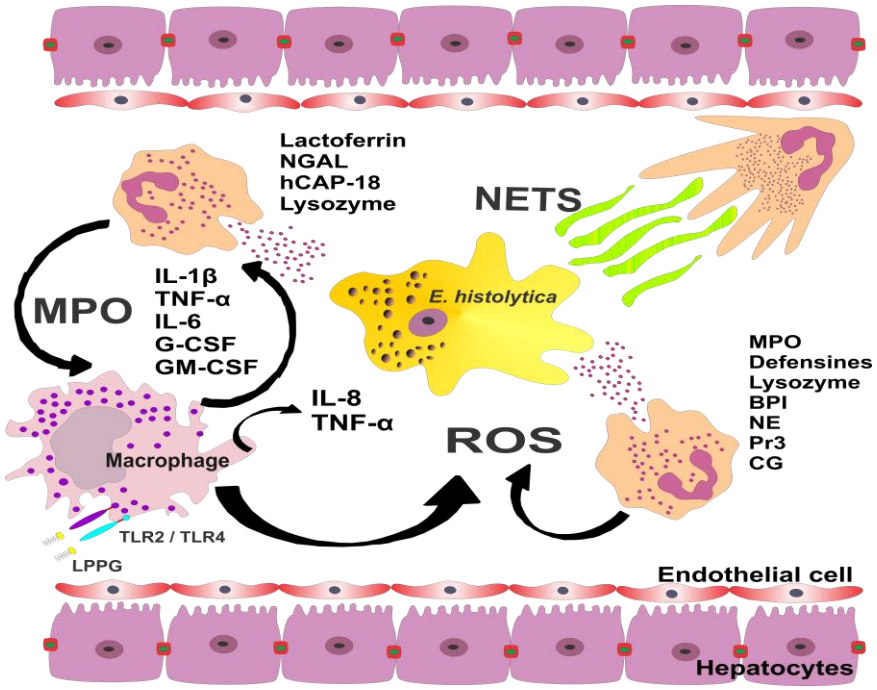
Host factors

The human host represents the major reservoir. Interpersonal transmission occurs via files and handles, and by sewage contamination of water sources. Male homosexuals harbor non- pathogenic E. dispar. A high iron content and carbohydrate rich diet.These predisposes to invasive amoebiasis.

Pathogenesis

Three virulence factors arelectin,amoebapores and cysteineproteases . A diffuse mucosal damage before amoebic invasion. An amorphous, granular, eosionophilic material surrounds trophozoites in tissue.Inflammatory cells are found at the periphery of amoebic lesions.

Amoebiasis causes necrotic abscess or periportal fibrosis .The abscess contains cellular, proteinaceous debris .It is surrounded by a rim of amoebic Trophozoite invading tissue. Areas of hepatic necrosis, due to ischemia from amoebic obstruction of portal vessels . Amoebic liver abscesses result from the coalescence of micro abscesses.



The E. histolytica gatactose specific adhesion isolated by Petri et al. The adhesin is a 260 -KID surface protein that consists of I7OKD and 35KD subunits. The heavy subunit may mediate attachment as it is recognized by adherence- inhibitory monoclonal antibodies. Direct galactose binding activity of recombinant heavy subunit produced by expression DCR methodology has been demonstrated.

The heavy and light subunits are encoded by gene families. The heavy subunit has a short cytoplasmic domain, a transmembrane domain, and a large extra cellular portion with a distinct cysteine- rich area.

The light subunit in contrast is attached to the membrane via a glycosyl-phosphatidylinositol anchor. Petri et a1 identified seven discrete epitopes in the heavy subunit using monoclonal Ig antibodies all of which are located in the cysteine-rich domain.

Entamoeba histolytica contains numerous proteolytic enzymes, including a cathepsin, proteinase, an acidic proteinase, collagenase and a well characterized major neutral proteinase.

Clinical features:

Amoebic liver abscess 90% occurs in young adult male. A history of international travel by the patient to his or her close contacts may be relevant. History of homosexual activity should be asked. A history of previous dysentery is infrequent . Symptoms of amoebic liver abscess are slow in onset and present for several days or weeks before medical attention is sought. Initial complaints are vague and include malaise, fever, anorexia and abdominal discomfort. Right hypochondrium Pain is most often the dominant symptom . About 3/4th of patients complain of fever, often with chills at night. Anorexia, nausea and vomiting are present. Chest symptoms are present in about 1/4th of patients include right-sided pleuritic pain and cough. Diaphragmatic irritation may result in right shoulder pain and hiccoughs . Patients may recognize abdominal swelling. Concurrent dysentery or diarrhea, is rare.

	% of Amebic Abscesses
Symptom	
Pain	90
Fever	87
Nausea and vomiting	85
Anorexia	50
Weight loss	45
Malaise	25
Diarrhea	25
Cough or pleurisy	25
Pruritus	<1

On examination Fever and right hypochondrial tenderness present.Hepatomegaly give the abdomen an asymmetrical appearance.

Most often the liver is palpable. The physical signs may be subtle when the abscess is in the left lobe of the liver. Presence of epigastric and left hypochondrial tenderness may arouse suspicion of enlargement of the left lobe of liver.

Sign	
Hepatomegaly	85
Right upper quadrant tenderness	84
Pleural effusion or rub	40
Right upper quadrant mass	12
Ascites	10
Jaundice	5

In 50% cases restriction of the right chest movement may be limited by pain. Dull on percussion over the right lower lung field is common .It shows a raised right hemidiaphragm or pleural effusion. Rarely fine creptations on auscultation or a pleural or pericardial friction rub present.

Jaundice when present indicates severe illness. Deeper jaundice usually results from multiple or large amoebic abscesses .Also from lesion situated near the inferior surface of the liver due to compression of the larger intrahepatic ducts.

Most cases of liver abscess in childhood have been in children under age 3, with some affected at only 1 month of life. The sex ratio of cases in children is almost equal. Fever and tender hepatomegaly are the usual physical signs. Associated intestinal amoebiasis and multiple hepatic abscesses seem more frequent .

When liver abscess occur in pregnancy, frequently such cases are misdiagnosed.The immunologic and hormonal alterations of pregnancy predispose to invasive disease. Amoebic liver abscesses is rare in patients with chronic liver disease.

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DIAGNOSIS

Anaemia is common in amoebic liver abscess. A neutrophilic leucocytosis with high proportion of bands may be seen. Although the white blood cell count is between 10,000 and 20,000/ul isolated cases with leukemoid reactions are described. The erythrocyte sedimentation rate is raised.Reduction of serum albumin levels are the most frequent abnormal.

Laboratory data	
Increased alkaline phosphatase	80
WBC count >10,000/mm^3	70
Hematocrit <36%	49
Albumin <3 g/dL	44
Bilirubin >2 mg/dL	10

Diagnosis of liver abscess is confirmed by a positive serologic test.It is highly sensitive (>94%) and highly specific (>95%)

Radiological investigations:

About 50% patients show elevation of the right hemi diaphragm of the X-ray chest PA view. Blunting of the right costophrenic angle from a sympathetic pleural effusion is common..

Technetium sulphur colloid scanning the first modality that allows direct assessment of space occupying liver lesions is sensitive but lacks specificity. Other hepatic masses, such its as tumors and cysts, produce "Cold' Areas. Gallium scans used to complement sulphur colloid scans. Unlike pyogenic abcesses and primary hepatocellular cancers, amoebic abscesses concentrate gallium only at the periphery of the abscess. The disadvantages of these tests include their low specificity.

Ultrasonogram is fast, safe, economical, and easily repeatable. Its disadvantage is operator dependency.Ultrasonic signs mentioned as typical of hepatic amoebic abscess are 1) oval or round shape 2) a lack of notable wall echoes, so that there is abrupt transition from normal liver to the lesion 3) a hypoechoic appearance compared with normal liver 4) a peripheral location close to the liver capsule: and 5) a distal sonic enhancement. A typical features that have been documented include on irregular shape and a hyper echoic appearance

LIVER ABSCESS – ULTRASONOGRAM

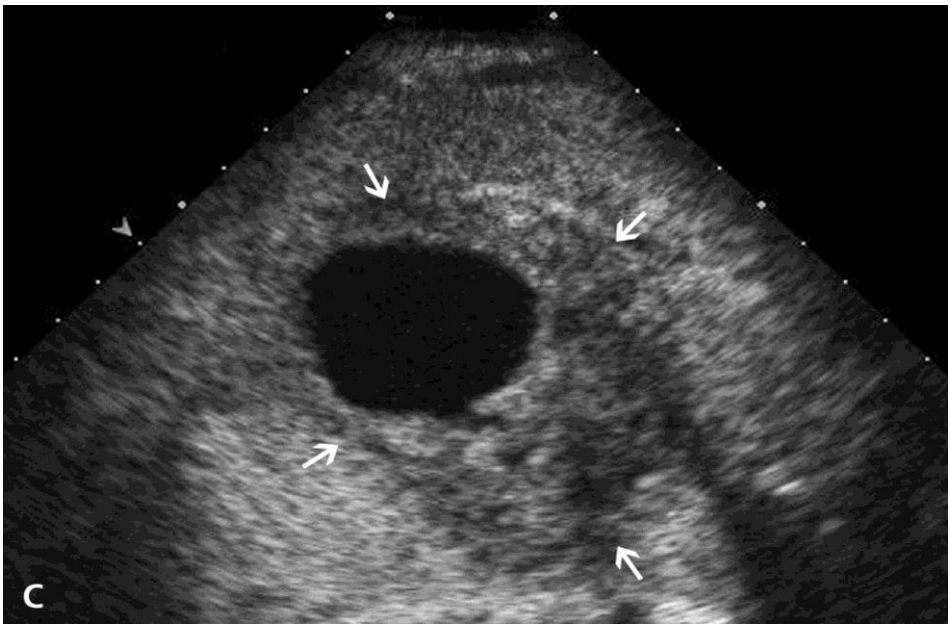
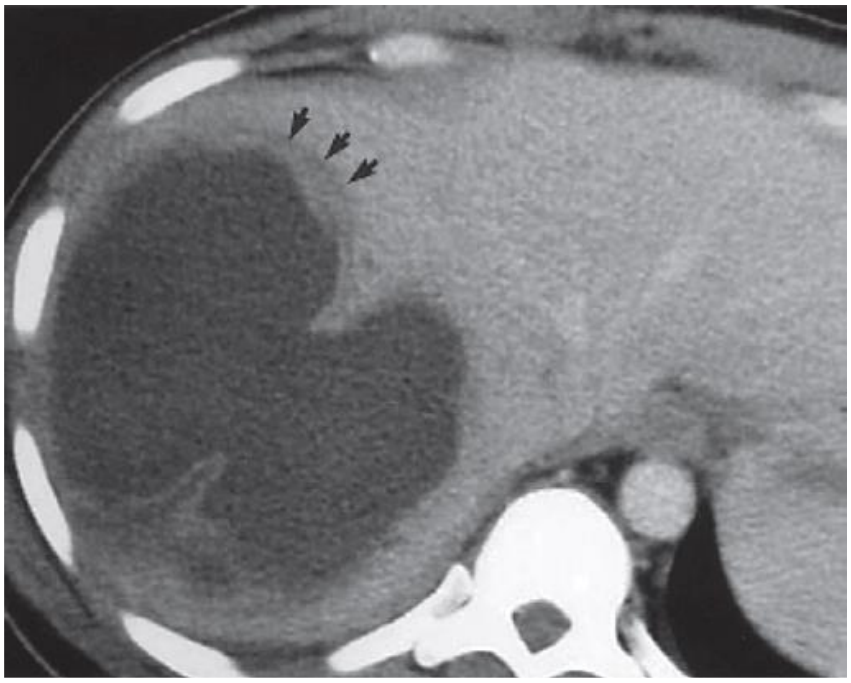


Table 3. Ultrasonographic distribution in both groups of liver abscess.

No. of abscess	Group A (amoebic)		Group B (Pyogenic)	
	No.	%	No.	%
Single	36	78	0	0
Multiple	10	22	6	100 (P=0.0004)
Location				
Right lobe	36	78	3	50
Left lobe	7	15	1	17
Both lobes	3	7	2	33

Computed tomographic scanning shows amoebic abscesses well defined, round, low density lesions, which may have a non homogenous internal structure. CT scanning is particularly useful in precise localization and definition of extent of disease.



Serodiagnosis:

Stool examination in amoebic abscesses patient have been negative in 3/4th cases. Over diagnosis is especially common, with stools leucocytes frequently reported as trophozoites of E. histolytica

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Serodiagnostic tests used include complement fixation, immunodiffusion, indirect fluoresent antibody tests, IHA, Counterimmuno electrophoresis, and ELISA. Diagnostic kits using latex agglutination are available.

The IHA test is highly sensitive and widely available. A serologic titer of 1:512 is usual, although not invariable, in acute invasive disease. Titres may continue to rise after presentation, and on occasion, the test is negative when the patient is first seen but positive a few days later. The IHA test may remain positive for months or years after invasive infection. ELISA is a cheap and sensitive technique that has been widely applied to the serodiagnosis and seroepidemiologic study of many parasitic diseases. Its use for the diagnosis of amebiasis is likely to increase.

Role of PCR

Nested PCR and multiple PCR are helpful in differentiations the various species of entameba i.e. E.histolytica .E. dispar., E.meshkorskii.

Role of Diagnostic aspiration:

They are done if amoebic serology are negative. Aspiration of anchovy sauce pus from the confirm the diagnosis of amoebic liver abscess. Nowadays, USG-guided aspiration is often justified .Fluid of amoebic abscess are odourless .It is also gram stain negative.Amoboea can be recovered in 33-90% cases. The routine aspiration of uncomplicated amoebic liver abscess not recommended. Two recent studies have shown that aspiration does not accelerate healing.This may only confuse the diagnosis by revealing atypical pus or blood. This approach is supported by a recent small prospective study. Clinical improvement invariably occurs with antiamoebic therapy alone in an uncomplicated case.

Aspiration is therefore now regarded as generally superfluous in the management of amoebic liver abscess, and should be reserved for situation when

1) Amoebic serology is inconclusive, delayed, or unavailable and the main differential diagnosis is a pyogenic liver abscess.

2)A therapeutic trial with antiamoebic drugs is deemed inappropriate

3)There is suspicion of secondary infection of the liver abscess.

4)When fever and pain persist for more than 3 to 5 days after starting appropriate therapy, aspiration may provide symptomatic relief.

TREATMENT OF LIVER ABSCESS:

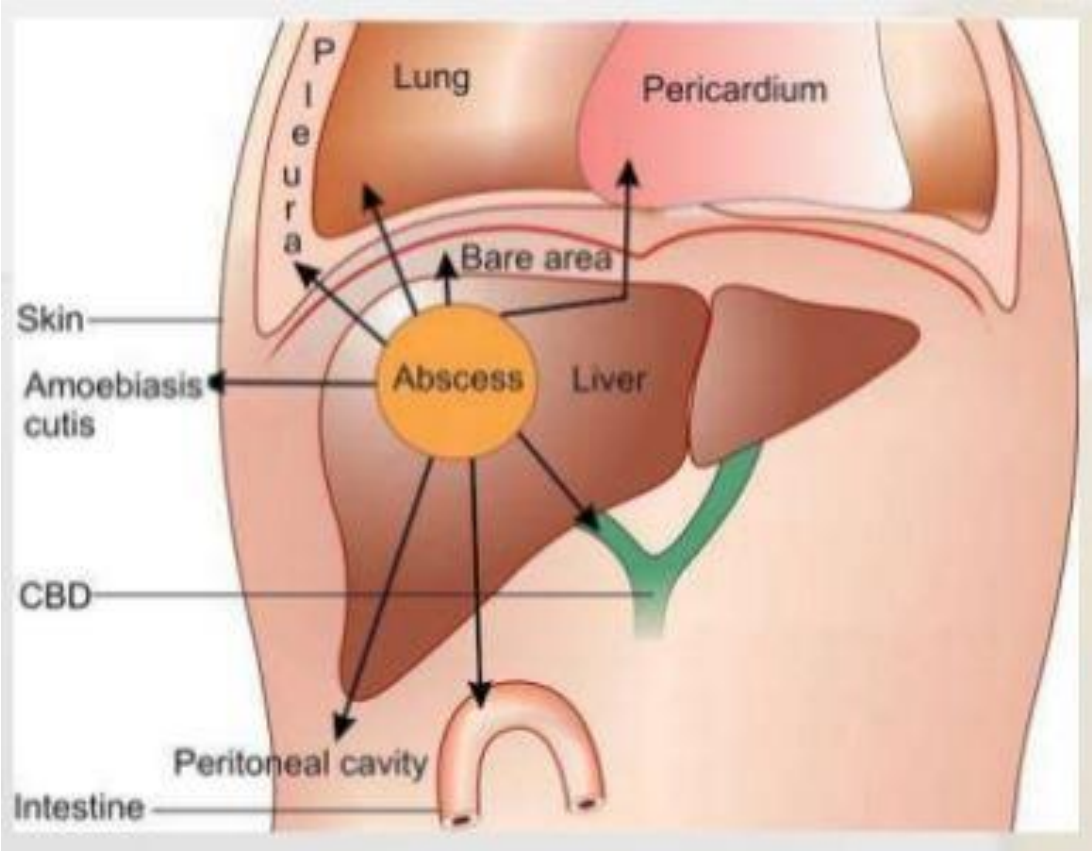
TABLE 118-5 Treatment of Amebic Liver Abscess

Drug treatment	Uncomplicated amebic hepatic abscess
	Both amebic colitis and liver abscess—nitroimidazole derivatives (e.g., metronidazole)
	Amebic colitis—luminal agents such as paromomycin, diloxanide furoate, iodoquinol
Percutaneous drainage	Deterioration in clinical condition despite adequate treatment
	Bacterial superinfection
	Abscess with high risk of rupture
Surgery	Ruptured abscess
	Impending rupture
	Inadequate catheter drainage

Drug	Adult dosage
Metronidazole	750 mg tid × 7–10 days
–or–	
Tinidazole	2 g/d divided tid × 3 days
–followed by–	
Iodoquinol	650 mg tid × 20 days
–or–	
Paromomycin	25–35 mg/kg/d divided tid × 7 days
–or–	
Diloxanide furoate	500 mg tid × 10 days

Complications of amoebic liver abscess:

Amoebic liver abscesses rupture into neighboring cavities and organs — the peritoneum, viscera and large vessels on one side of the diaphragm and the pleura, bronchi, lungs and pericardium on the other.



Peritoneal and visceral involvement:

Peritonitis associated with amebiasis is due to a rupture of amoebic liver abscess in 78% of cases and due to perforated or necrotizing amoebic colitis in 22%.

The incidence of spontaneous rupture of amoebic liver abscess varies between 2.7 and 17% of cases. Between 18 and 70% of all amoebic liver abscess ruptures are into the peritoneal cavity. Adherence of the liver abscess to the diaphragm, anterior abdominal wall, omentum and bowel tends to confine the area of contamination. Free rupture into the peritoneal cavity is uncommon .It occurs in a nutritionally depleted and moribund patient. Patients present with abdominal pain and a mass or generalized distention. Sudden bloody diarrhea may occur in colonic rupture . Hematemesis may occur in patients with hepatogastric fistula. Signs of peritonitis along with tender hepatomegaly, intercostals tenderness and right basal lung signs and clinical jaundice seen. When diagnosis may be made only at laparotomy, at which time the excessive bleeding resulting from decreased prothrombin levels can be difficult to manage.

USG and CT often show perihepatic fluid collection in cases of amoebic liver abscess.

Absolute indications for laparotomy include 1)doubtful diagnosis, 2)concomitant hollow viscus perforation, or 3) if conservative management fails. At laparotomy the liver abscess, which usually appear as a tan-colored bulge on the surface.. Septa running across the cavity are usually blood vessels and bile ducts . Hemorrhage can be difficult to control and postoperative bile leaks may result. Irrigation of the abscess cavity with saline is usually sufficient and may be followed by the installation for 3-5 mm of a solution of 65 mg of emetine hydrochloride in 100 ml of normal saline. Tube drains are inserted and retained as necessary.

Postoperative antiamoebic therapy in the form of intravenous metronidazole is combined with broad-spectrum antibiotics. Dehydroemetine is added if no cardiac contraindication exists.

Thoracic and pleuropulmonary involvement

Rupture of the abscess into the pleural cavity and rupture of abscess into the bronchial tree can occur.

Transdiaphragmatic involvement in abscess located high on the right lobe. Presents with dyspnea and a dry cough which exacerbates the right hypochondrial pain caused by the hepatic lesion. Right basal crepitations are a frequent . A pleural rub can be heard. CXR shows atelectasis and blunting of the costophrenic angle. USG and CT identifies the pleural effusion before clinically detectable.Thoracocentesis is required.

Rupture of the abscess into the bronchi is characterized by the sudden onset of coughing with expression of copious quantities of chocolate-colored sputum. Metronidazole used as a single drug is effective in the treatment of thoracic complications of amoebic liver abscess, but emetine produces a more rapid response and may be required in cases where metronidazole resistance occurs

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Chemotherapeutic agent

Metronidazole : is the treatment of choice for all forms of invasive amoebiasis. It is a nitroimidazole that is well absorbed after oral administration, and it is excreted mainly by way of kidneys.

Adverse effects include nausea,. anorexia, metallic taste, dark urine and a disulfiram like reaction with alcohol. Central nervous system effects such as vertigo, ataxia, and peripheral neuropathy have also been reported.

The usual dosage of metronidazole is 800 mg three times daily for 5 to 10 days. The usual paediatric dose is 35 to 50 mg/kg/d in three divided doses

Chloroquine: The antimalarial drug chloroquine, a 4- aminoquinoline, acts by binding to parasite deoxyribonucleic acid. . The usual dose is 1 gm / day for 2 days followed by 500 mg/day for 20 days. The only controlled trail of chloroquine versus metronidazole for amoebic liver abscess showed no difference in efficacy

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Emetine and dehydroemetine

Emetine is most potent amoebicidal drug . It is given by i.m or s.c injection .It is excreted through the kidneys. It interfers with protein synthesis. The usual dosage is I mg/kg/d to a maximum of 60 mg/day for 10 days. Adverse effects include vomiting, diarrhoea, renal impairment, and pain or necrosis at the site of injection. The most serious adverse effect is cardiotoxicity.

Dehydroemetine is a synthetic preparation .It has a similar action to emetine with less cardiotoxicity. It is equally effective therapeutically but excreted more rapidly. The daily dose of 1.25 mg/kg is given by im or s.c injection to a maximum of 90 mg/d.

Therapeutic strategy:

Metronidazole is administered as a single drug after diagnosis, with concomitant correction of hypoprothrombinemia, hypoproteinemia, and anaemia. If improvement in 48 - 72 hours is present no other therapy needed. A luminal agent such as Dilonaxide furoate (500 mgm p.o. tid x 10 days) or paromomycin (30 mg/kg/day in 3 days x 10 days) must be given to complete treatment.

Evidence of pulmonary, peritoneal or pericardial extension is all indication for aspiration of the liver abscess with an intercostal tube or catheter drainage into a closed-circuit collection system. Failure to adequately control the abscess constitutes as indications for Laparatomy.

PROGNOSIS

Meta-analysis of 3081 patients with amoebic liver abscess showed that 114 (4%) died. In comparison, the mortality rate for pyogenic liver abscess was

46%. In patients treated with amoebicidal drugs alone the mortality was 2% and the addition of needle aspiration did not improve this result. Independent risk factors for mortality include serum bilirubin more than 3.5 mg%, encephalopathy, hypoalbuminemia less than 2.0 G% and multiple abscess cavities.

Ruptured amoebic liver abscess occurs in 2-17% of patients, with mortality between 6 and 50%. It is hoped that with increasing skill at percutaneous drainage of these abscesses the mortality in these patients, who usually constitute a major risk for surgery and anesthesia, will be reduced

STUDY PROTOCOL

This will be a CLINICAL PROSPECTIVE STUDY of 60 patients done at KMCH between January to September 2016.

METHODS OF COLLECTION OF DATA (INCLUDING SAMPLING PROCEDURE):

A. Study Design: Prospective cohort study.

B. Place of study: Govt. Kilpauk Medical College and Hospital, Chennai.

C. Study sample size: $N = Z^2 P\{1-P\} / d2 = 60$ with 95% confidence interval z value is taken as 1.96

P= Propotion of people with amoebic liver abscess/alcoholism 70 %

D= absolute error 12%

So applying these variables in the formula sample size is 60.

SAMPLE SIZE : 60 (selected by Random sampling method)

D. Study period : 9 months (January to September)

E. Selection criteria :

First 60 patients admitted during the period of study.

MATERIAL AND METHODS

SOURCE OF STUDY

All patients of Liver Abscess presenting to the Surgery OPD or Casualty of RoyapettahHospital, referred from medical wards of Royapettah hospital or referred from outside diagnosed as case of liver abscess Clinical/ Sonological/CT/MRI features of Liver Abscess

INCLUSION CRITERIA:

All cases of liver abscess diagnosed clinically and/or ultrasonographically.

EXCLUSION CRITERIA:

- Traumatic Liver Abscess
- Past history of liver abscess

METHOD OF COLLECTION OF DATA:

- 60 eligible patients are chosen.
- Clinical assessment done at time of inclusion in the study.
- Detailed history and examination done.
- Diagnosis to be confirmed by ultra-sonogram of abdomen.
- Pus drained will be sent for culture and sensitivity appropriate antibiotic coverage will be given.

- Basic routine investigations and coagulation profile will be done.
- Consent will be obtained for inclusion under study
- Patients will be followed up daily clinically and LFT & USG Abdomen will be repeated on the 3rd day if patient is symptomatically not relieved.
- Repeat Ultrasound / CT /MRI Abdomen & pelvis will be done immediately if patients condition does not improve/worsens or after 3-4 days as a routine as a prognostic factor.
- If the patient develops any of the complications like ruptured liver abscess into any of the serosal cavity , will be immediately taken up for surgery.
- Patient informed about any surgical procedure and consent will be obtained.

DIAGNOSTIC CRITERIA

All the patients had several investigations required to approach the diagnosis and they were diagnosed as amoebic or pyogenic liver abscess. Basically USG abdomen, serology and pus c/s were done. Serology positive and USG characteristics of smooth wall homogenous with no internal echoes and superficial solitary abscess were grouped as amoebic .

Serology negative and pus c/s negative cases with USG characteristics of amoebic abscess were also considered as amoebic abscess

LUNG INVOLVEMENT

X—ray chest PA view was taken in all cases. X—ray findings of right pleural effusion, presence or absence of cough with expectorations were considered as positive.

TREATMENT GIVEN:

Cases with abscess cavity < 5 cm were treated by drug therapy alone. Failures to relieve symptoms within 3 to 4 days were treated by percutaneous aspiration.

Those with abscess cavity > 5 cm were treated either by percutaneous aspiration or by percutaneous catheter drainage. Bilateral abscess cavities that were small and multiple were managed by medical therapy and when any one of the cavity is > 5 cm, it was managed by percutaneous aspiration.

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Abscess cavities restricted to left lobe were treated by drug therapy if they were multiple and < 5 cm ; if > 5 cm and single were managed either by percutaneous aspiration or by laproscopic drainage.

Those abscess cavities that were > 10 cm or with chances of impending rupture in segment III, IV, V, VI were managed by laproscopic drainage.

MEDICAL TREATMENT STRATEGY FOLLOWED WAS:

Abscess cavities that were < 5 cm were treated by Tab. Metronidazole 800 mg for 10 days.

Patient was on i.v metronidazole for three days initially or till the fever subsided. Later oral metronidazole is given and percutaneous aspiration was done and continued if patient had persisting symptoms after 3 to 4 days of aspiration.

PERCUTANEOUS ASPIRATION

Patient with abscess cavity > 5 cm were treated either by percutaneous aspiration or PCD. Multiple abscesses and the abscess fail to respond with medical treatment were percutaneously aspirated.

Uunder USG guidance it is done by using 16G or 1 8G aspiration needle or 3 way adopter as a single prick. First, aspiration was done followed by drugs. If symptoms are not decreasing after 3 days, do repeat USG and assess the cavity size.

If the cavity is increasing in size or not decreasing do 2^{nd} aspiration and continue drug therapy. If the symptoms are not subsided by 7th postaspiration day and USG showed the cavity is not decreasing or increasing in size, consider PCD or laproscopic drainage.

PERCUTANEOUS CATHETER DRAINAGE

PCD was done by using Malecots /22 F foleys under Ultrasonogram guidance with closed drainage system.

LAPAROTOMY FOR RUPTURED ABSCESS

PIGTAIL DRAINAGE

REMOVAL OF PCD

1. If the Quantity is less than 30 ml /8hrs.

2. IF the drainage is not purulent.

3. USG and cavitogram were done to assess the cavity size. Note down the decrease in size of the cavity and the PCD can be removed.

LAPROSCOPIC DRAINAGE

Patients with large abscess greater than 10cm and large abscess that was located in the left lobe of liver not amenable to percutaneous drainage were treated by laproscopic catheter drainage. Smaller 16 /14 F foleys used for abscess drainage and the same criteria of removal similar to that of PCD was employed.

RESULTS

The following observations were made in this study

Fig 1:DEMOGRAPHIC PROFILE

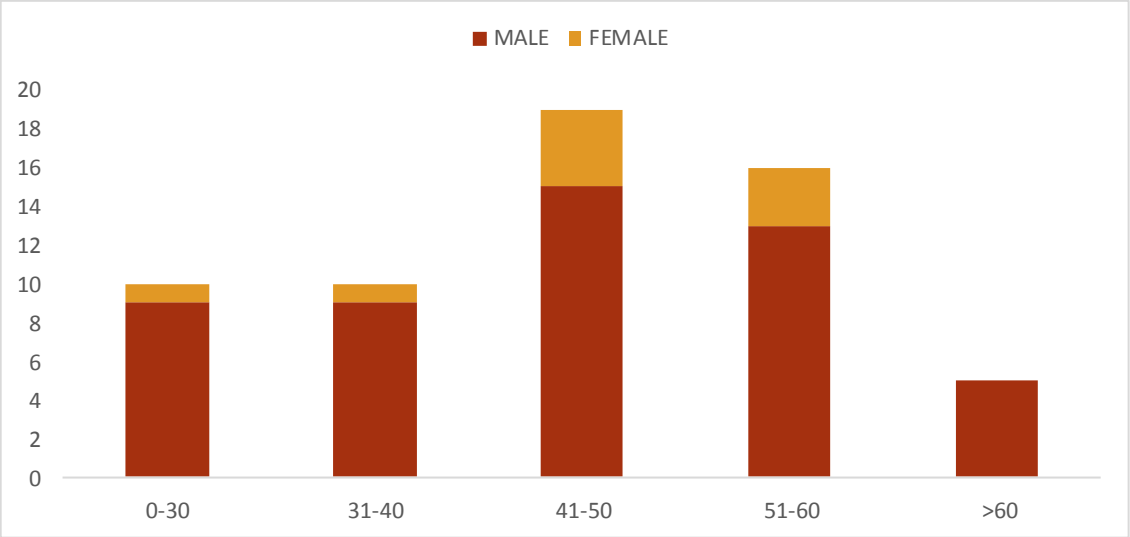


Table 1: AGE AND SEX DISTRIBUTION

Age Group	Male		Female		Total	
	No.	%	No.	%	No.	%
0-30	9	17.64	1	11.11	10	16.66
31-40	9	17.64	1	11.11	10	16.66
41-50	15	29.4	4	44.44	19	31.66
51-60	13	25.49	3	33.33	16	26.66
61 yrs	5	9.8	0	0	5	8.33
Total	51	100	9	100	60	100

The mean age distribution of the study group is 45.34 with youngest patient at 21 years of age and oldest patient being 66 years of age.It is more common in males (85%) than females (15%). The commonest age group for liver abscess was 41-50 yrs (31.66%) followed by 51-60 (26.66%).

Table 2: PRESENTING SYMPTOMS

Symptoms	No. of patient	%
Fever	45	75
Abdomen pain	44	73.33
Jaundice	27	45
Cough	2	3.33
Diarrhoea	8	13.33
Altered sensorium	1	1.66

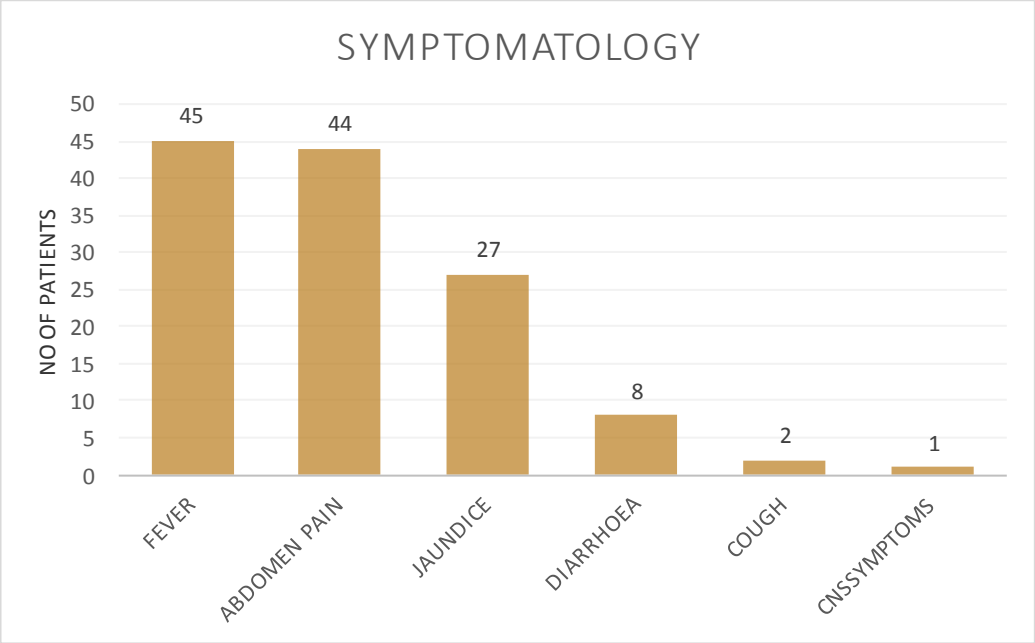


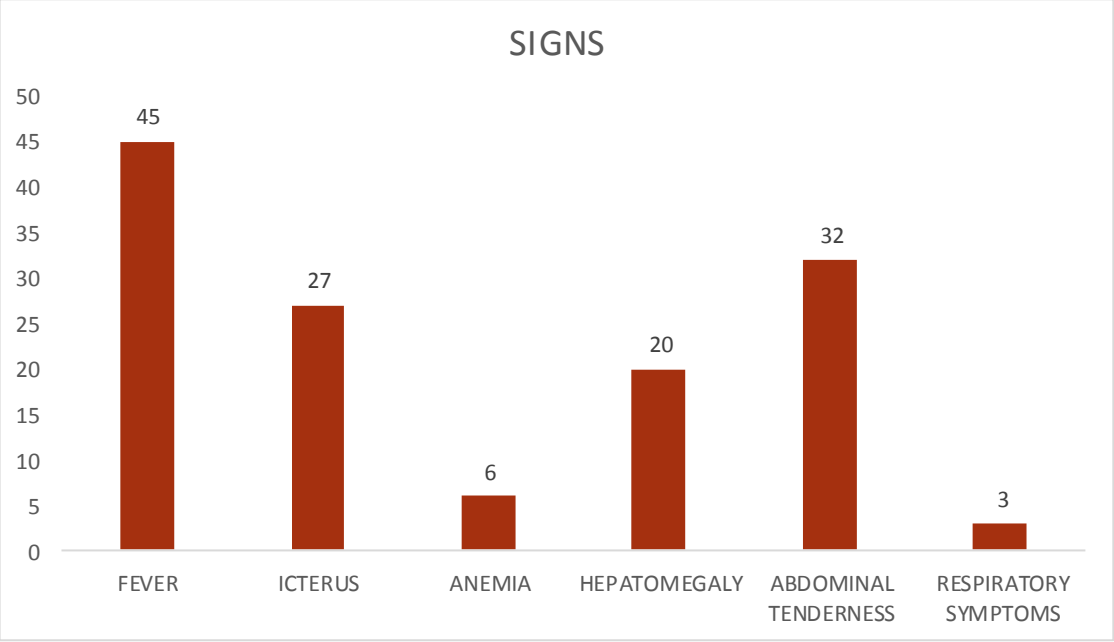
Fig 2: PRESENTING SYMPTOMS

The commonest symptom was fever(75%) then by abdomen pain (73.33%), Jaundice was present in 45% ,diarrhea occurring in 13.33%, cough in 3.33% and altered sensorium 1.66%.

Table 3: SIGNS

Signs	No. of Patient	%
Fever	45	75
Icterus	27	45
Pallor	6	10
Hepatomegaly	20	33.33
Abdominal tenderness	32	40
Respiratory findings	3	3.75

Fig 3: SIGNS

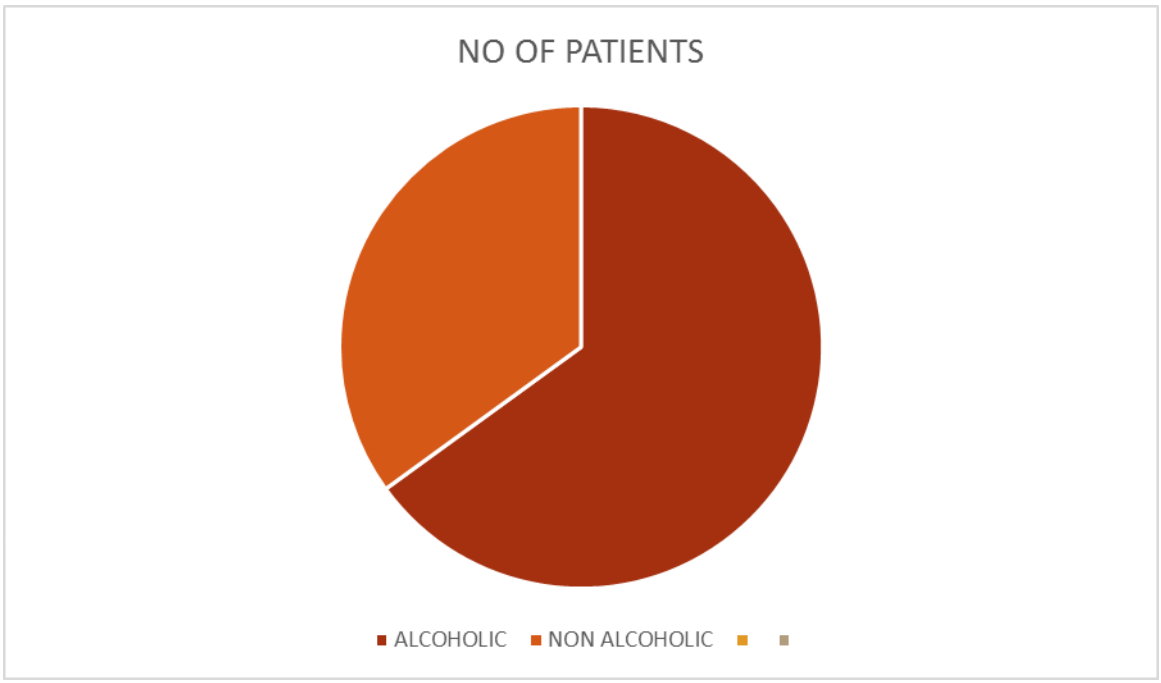


The most common sign was fever which was present in 75% patients, 40% of patients had abdominal tenderness at the time of diagnosis and 32.5% patients had hepatomegaly, 45% of patients had icterus, pallor was present in 10% of patients and respiratory findings in 3.75% of patients which include right pleural effusion, basal crepitations.

Table 4: DURATION OF STAY (MORBITIY)

Onset	No. of patient	%
Short duration <10 days	29	48.33
Long duration	31	51.66

In this study patients duration of stay in hospital from admission till 10 days was 48.33% of cases and >10 days is 51.66%


NO OF PATIENTS
ALCOHOLIC
NON ALCOHOLIC

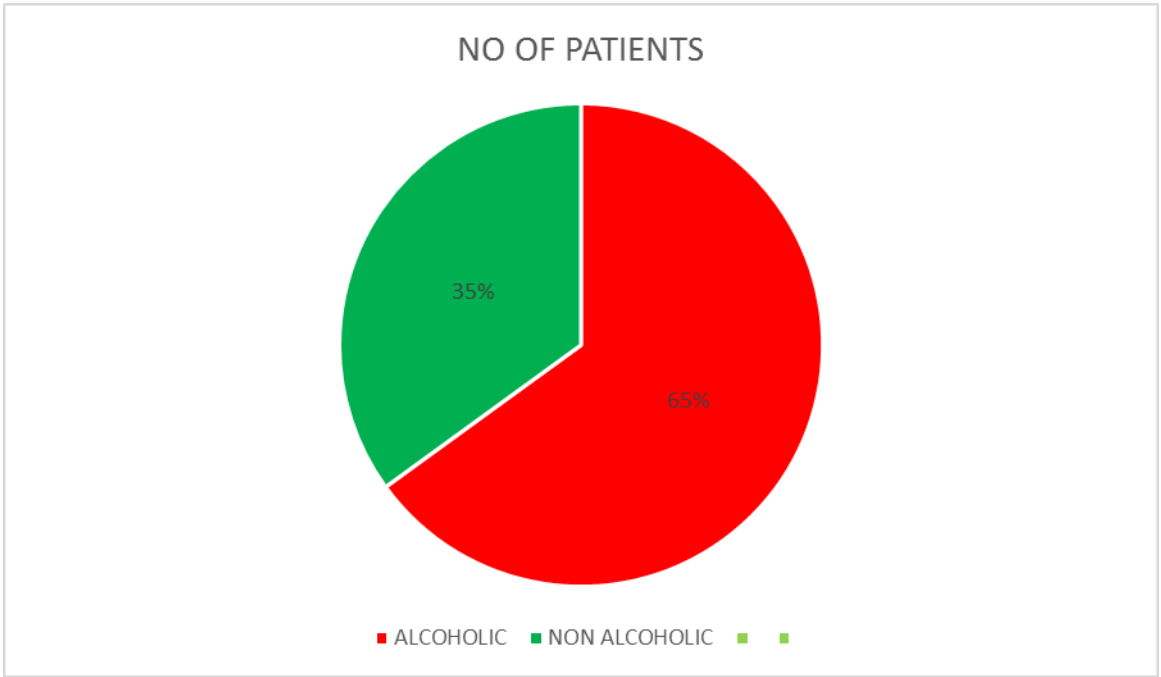

NO OF PATIENTS
35%
65%
ALCOHOLIC
NON ALCOHOLIC

Table 6: LAB PARAMETERS

Investigation	**No. of patient**	**%**
Anaemia (Hb < 10 gm%)	6	10
Leucocytosis (> 12,000 c/cmm)	41	68.33
Diabetic (RBS > 200 mg/ dl)	12	20
Raised urea (> 60 mg / dl)	7	11.66

Hemoglobin less than 10gm% was found in 6 cases (10%) , lowest hemoglobin noted in this series was 7.6 gm%.

Leucocytosis of more than 12,000 cells / cumm was present in 41 patients (68.33%). The highest count noted in this study was 22,000 cells / cumm.

1/5TH (20%) of patients were found to be diabetic.

Raised urea (> 60mg/dl) was found in 7 cases (11.66%).

Table 7: ABNORMAL LFT

	NO OF PATIENTS	PERCENTAGE
HYPOALBUMINEMIA	6	10%
HYPERBILIRUBINEMIA	33	55%
RAISED ALP	31	51.66%
ABNORMAL PROTHROMBIN TIME	4	6.6%

Fig 5 : BILIRUBIN LEVELS

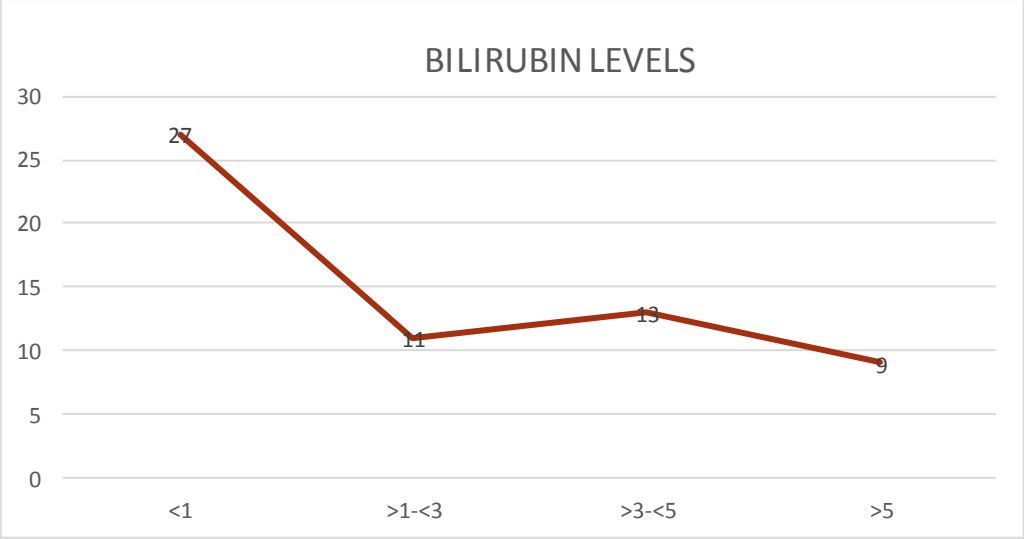


Table 8: ANATOMICAL LOCATION OF ABSCESS

Ultrasonogram examination was done in all cases.

Location	No. of patient	%
Right lobe	47	78.33
Left lobe	6	10
Both lobes	7	11.66
Total	60	100

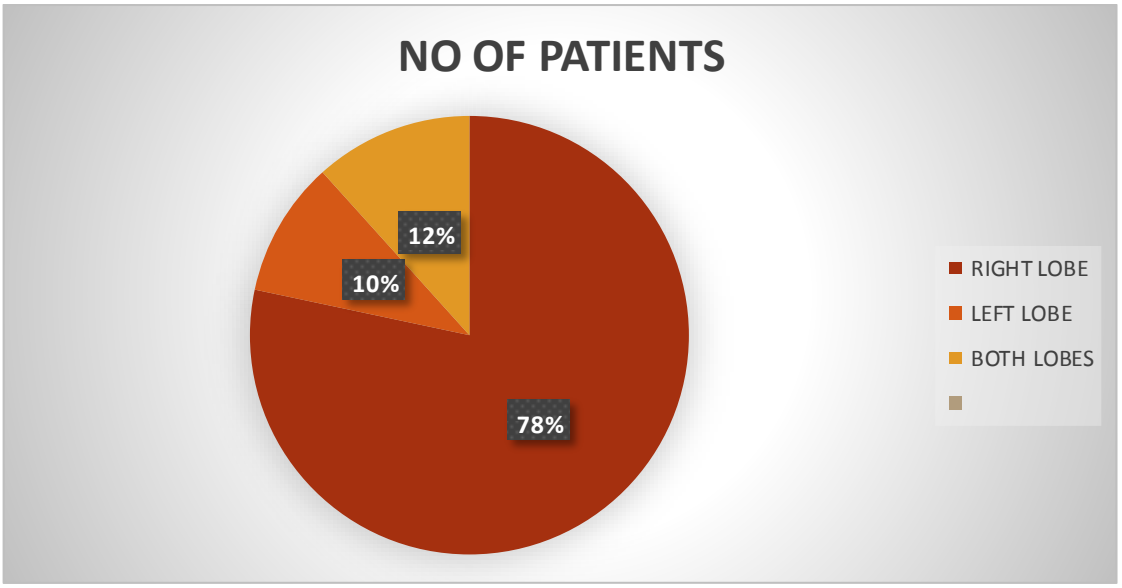
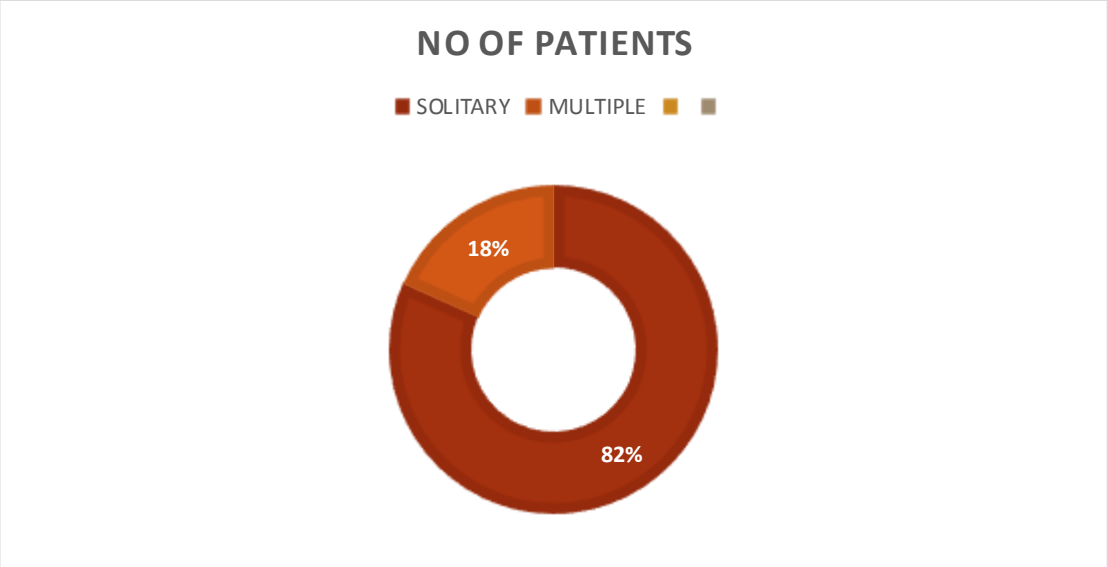


Fig 6: ANATOMICAL LOCATION OF ABSCESS

Fig 7: SOLITARY / MULTIPLE ABSCESS

Table 9: SOLITARY / MULTIPLE ABSCESS



Number	No. of patient	%
Solitary	49	81.66
Multiple	11	18.33
Total	60	100

Table 10: PUS CULTURE ANALYSIS

Organism	No.of patient	%
No growth / Anchovy sauce	36	87.8%
Gram –ve	3	7.31%
Staph aureus	2	4.87%
Total	41	100%

In this study 41 cases were subjected to invasive treatment. Out of 41 cases, 36(87.8%) had "Anchovy sauce" appearance of the pus and revealed no growth. While growths were obtained in 5 (12.1%) of these cases, gram – ve organisms grown in 3cases (7.3%) and staph aureus in 2 cases (4.8%).

Table 11: TREATMENT ANALYSIS

Treatment	No. of patient	%
Conservative	19	31.66
Aspiration/pigtail drainage	31	35
Laparatomy	5	8.33
Laparoscopic abscess drainage	5	8.33
Total	60	100

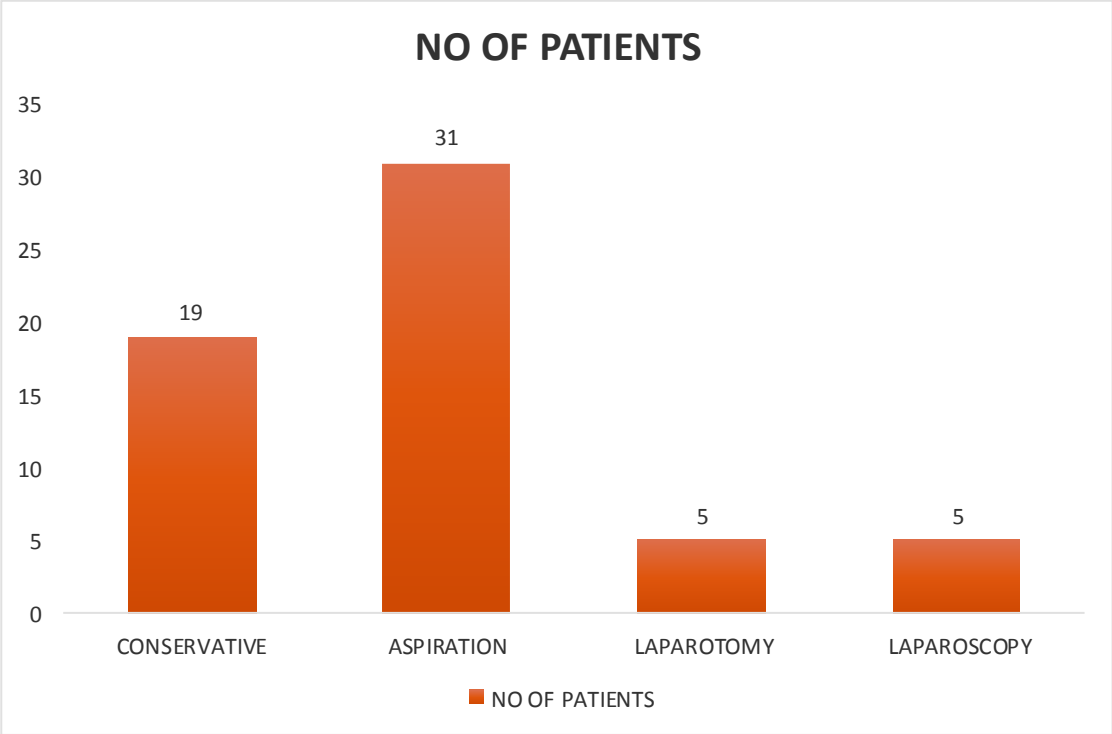


Fig. 8: Treatment of liver abscess

Of 60 cases, with liver abscess, the volume is < 50 cc is 19 cases (31.66%) were treated conservatively and those with volume > 50cc were treated by USG guided aspiration is 31 cases (51.66%). 5 cases were treated by Laparatomy and 5 cases by laparoscopic abscess drainage. Abscess ruptured into peritoneal cavity in 5 cases hence laparotomy done. Pus was completely drained out and sent for microbiological examination, peritoneal toilet was given.

Table 12: COMPLICATIONS

Complications	**No.of patient**	**%**
Ruptured into peritoneal cavity	5	8.33
Pleural effusion	2	3.33
Septicemia	1	1.66

Table 13: MORTALITY RATE

Total patient with liver abscess	60
Surviving	59
Death due to liver abscess	1

DISCUSSION

AGE AND SEX INCIDENCE

The age of the patients varied from 21 – 66 years.. The mean age was 45.34 yrs which is in accordance to studies like by Sharma et al and Mukhopadhyay et al who reported it to be 40.5 and 43.64 years, respectively. The highest incidence was noted in the age group 41-50 years of age (31. 66%) followed by 51-60 years of age (26.66%) yrs in this study

Indian data show predominant male involvement; Sharma et al. and Mukhopadhyay et al. reported male to female ratio to be 7 : 1 and 11 : 1, respectively. However, Pang et al. and Heneghan et al. reported it to be 2 : 1 and 1.22 : 1, respectively

Table 14: The comparison of symptoms and signs in present study with literature

SYMPTOMS	**STUDY**	**Sharma et al**
	No of cases (%)	**%**
Fever	45 (75)	94
Pain abdomen	44 (73.3)	90.6
Jaundice	27 (45)	12.7
Cough	2 (3.33)	3.5
SIGNS		
Fever	45(75)	95
Icterus	27(45)	24
Abdominal Tenderness	32(53.33)	42
Hepatomegaly	20(33.33)	39
Respiratory finding	3(5)	37

The frequency of fever and pain abdomen is 67-87% and 62-94% of patients with amoebic liver abscess respectively in different series . In our study, these two symptoms of fever and pain abdomen occurred in 75% and 73.33% respectively . From India, Sharma et al in a study of 70 cases of amoebic liver abscess found hepatomegaly in 84%, pleural effusion in 10% and ascites in 4% cases . In contrast, hepatomegaly (33.33%) was not a predominant feature of amoebic liver abscess in our study

Duration of symptoms

The onset of the disease is subjected to great variations depending upon the type, location and quantity of liver abscess; it may be acute, insidious, clinically undetectable or fulminant form. In this present study acute onset <10days was seen in 48.33months and 51.66% with the chronic presentation of liver abscess.

Duration of symptoms longer than 2 weeks is seen in 14-41% in different series . In a study of amoebic liver abscess by Amarapurkar and colleagues of 131 patients, the duration of symptoms less than 2 weeks was seen in 83.9% of cases.

According to Maingot's abdominal operations, most patients of liver abscess manifest symptoms for less than 2 weeks but a more indolent course occurs in 1/3rd of the patients.

Alcoholism in cases of liver abscess

Alcoholism was found to be the most consistent etiological factor in this study of liver abscess. 65% of the cases of this study were found to be alcoholics. The presence of alcoholism as a risk factor was noticed in many studies. In Indian culture almost all the alcoholics are males. The age predisposition and gender differences may be as a result of high alcohol intake by young male which predisposes to ALA. Alcohol suppresses function of Kupffer cells (specialized macrophage) in liver which has important role in clearing amoeba. Moreover, invasive amoebiasis appears to be dependent on the availability of free iron. A high content of iron in the diet, often obtained from the country liquor in habitual drinkers predisposes to invasive amoebiasis, as does a diet rich in carbohydrate

Analysis of laboratory investigations:

10% of patients were found to be anaemic (Hb < 10gm /dl) in our present study. The mean Hb of the patients in this study was 10.4 gm/dl with a range 8.8-13.6 gm%.. According to Bhagwan satiani and Eugene D. Davidson, anaemia was present in 39% of cases[38]. There is less literary evidence suggesting anaemia is a predisposing factor for liver abscess. But

high incidence of anaemia is noted in many of the cases, and the relation is not well understood. Leukocytosis was observed in our cases (68.33%)which was comparable to other studies.

Diabetes Mellitus was observed in 20% of patients. The increased association of diabetic state with liver abscess shows that diabetes is a risk factor for liver abscess. According to A.J. Greenstein, D Lowenthal, BA, G.S. Hammer, F. Schaffner and A. H. Aufses, Diabetes was found in 10% of cases.

From India, earlier series showed jaundice in 45%-50% of cases of amoebic liver abscess, whereas in our study the elevated bilirubin levels were noted in 33 patients(55%).Pathogenic processes proposed which can lead to jaundice are sepsis, alcoholic liver disease, hepatocellular dysfunction, associated hepatitis in the adjoining areas, intrahepatic biliary obstruction by the expanding abscess, and biliovascular fistula resulting from hepatic necrosis leading to damage of bile ducts and hepatic veins

Raised ALP levels were noted in 51.66% of patients and observations by Bhagwan Satiani and Eugene D. Davidson increased levels of ALP was seen in 63% of cases[38]. According to Chu KM, Fan ST Hypoalbuminemia was an adverse prognostic factor in eases of liver abscess[44]. Increased prothrombin time > 20 was seen in 6.6% .

Table 17: USG Findings of liver Abscess

	Present study	sharma
	No of cases (%)	et al
Right lobe	78.33	71
Left lobe	10	17.5
Both lobes	11.66	11.5

Ultrasonogram abdomen was done to all patients in this study. In the present study right lobe was involved in 78.33% of cases. This is in accordance with the study concluded by sharma et al who recorded 71% involvement in right lobe.

The predilection of LA in right lobe is because of streaming effect in portal circulation . It receives most of blood draining from right colon, the primary site of intestinal amoebiasis.

TABLE18:DESCRIPTIVE STATISTICS

Descriptive Statistics

	N	Minimum	Maximum	Mean
Age	60	21	66	45.34
WBC	60	7000	22000	12599.17
hospital stay duration	60	3	42	12.17
Valid N (listwise)	60			

Null Hypothesis(H_0):

There is no significant difference between dependent variable Treatment and predictors – alcoholism, jaundice and abdominal pain.

Alternative Hypothesis(H_1)

There is significant difference between dependent variable Treatment and predictors – alcoholism, jaundice and abdominal pain

TABLE19:SUMMARY

Model Summary[b]

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.306[a]	.094	.040	1.083	.094	1.755	3	51	.168

a. Predictors: (Constant), alcoholism, jaundice, abdominal pain

b. Dependent Variable: Treatment

TABLE20:ANOVA

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	6.176	3	2.059	1.755	.168[b]
	Residual	59.824	51	1.173		
	Total	66.000	54			

a. Dependent Variable: Treatment

b. Predictors: (Constant), alcoholism, jaundice, abdominal pain

TABLE21:correlation

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	2.707	.389		6.964	.000
	abdominal pain	.541	.343	.220	1.576	.121
	jaundice	.443	.307	.201	1.442	.155
	alcoholism	-.448	.308	-.194	-1.454	.152

a. Dependent Variable: Treatment

In present study left lobe and both lobes were involved in 10% and 11.66 % of patients respectively where as 78.33% of patients had abscess cavity in the right lobe.

In the present study solitary abscess and multiple abscesses were present in 78.75% and 21.25% of cases respectively. This is in accordance with the study conducted by Chaturbhuj Lal Rajak et a1 who recorded 72% solitary and 18% multiple abscesses[45].

Pus culture analysis

41 cases in this study were subjected to invasive treatment out of which 87.8% cases had anchovy sauce appearance of pus and revealed no growth . gram (-) organisms found in 7.3% and Staph aureus found in 4%.

Analysis of treatment

In the present study of 60 cases patients who had multiple small abscess and solitary abscess with volume <50 ml were treated conservatively. The conservative management was done on 31.66% of cases.

All cases were started on metronidazole IV at a dose of 40 mg/kg/wt for 8-10 days. When patients did not show improvement in 24-48 hrs of metronidazole therapy, broad spectrum 3rd generation cephalosporins were started.

According to Hiroshi Okano, Katsuya Shraki percutaneous aspiration is not required in all cases of liver abscess. A subset of cases with small liver abscess < 300 cc can be successfully managed conservatively[46].

In 51.66% patients who had abscess >50 cc were chosen for percutaneous aspiration. The site, depth and direction of aspiration were marked under USG guidance, aspiration needle was usually used and under aseptic precautions, the abscess cavity was entered. Local anesthetic was used, pus was aspirated and sent for culture and sensitivity; no complication were noted due to this procedure apart from local pain which soon subsided after analgesics. Patient showed improvements in their symptoms and signs within 48-72 hrs of the aspiration. Percutaneous catheter drainage was not done on any patient in this study. Laparotomy as the initial line of treatment was performed in 5 (8.33%) of cases of liver abscess ruptured into peritoneal cavity. On laparotomy, thorough peritoneal lavage and drains were kept.

According to Arshed Zafar, Sajjad Ahnied, needle aspiration is safe, rapid effective method of treating liver abscess. Routine aspiration is not indicated. It should be initial line of treatment in abscess > 300 cc, impending rupture or abscess that do not respond to chemotherapy.

According to Antonia, Giorgio, Lucien Turuntino percutaneous needle aspiration is an efficient, effective and low cost technique that can even be performed on an out patient basis [48]. It is safe, free from significant complication.

Laparatomy was done in 6 cases for liver abscess which ruptured intraperitoneally. Laparoscopic liver drainage was done in 7 patients which is technically difficult for the young laparoscopic surgeons.

Discussion of complications

The complications in our study were rupture of liver abscess into peritoneal cavity and pleural effusion. Six case presented with peritonitis for which laparotomy was done and peritoneal lavage was given. Septicemia with multiorgan dysfunction was seen in 1 case. Pleural effusion was observed in 3 cases.

According to Sharma MP, Dasarthy S, Verma N et al, mortality rate in their study was 0-18 % and in our study one case (1.25%) had the end result of mortality due to liver abscess after intraperitoneal rupture

CONCLUSION

This study is based on the reports of 60 patients treated for liver abscess at Govt royapettah Hospital, kilpauk medical college,chennai.

- The most common age group affected by Liver abscess was between 41-50 years.

- The male-female sex ratio found in this study was 6:1.

- The most common symptom was fever, followed by pain abdomen.

- Alcohol consumption is important risk factor observed in the most number of patients.
- Solitary abscesses were common compared to multiple abscesses.

- The right lobe was more commonly affected.

- 2/3 of the patients needed invasive management.

- Most common cause is amoebic
- Multiple small abscesses and solitary abscess with volume less than 50 ml were managed successfully on conservative antimicrobial therapy alone.

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ANNEXURE

PROFORMA

Patient's Name : Address :

Age : Hospital :

Sex : Ward No :

Occupation : Unit :

Locality : Urban / Rural D.O.A :

Religion: D.O.D :

Duration of stay : Socio economic status:

CHIEF COMPLAINTS :

HISTORY OF PRESENTING ILLNESS:

Symptoms

1. Abdominal Pain : Yes / No

☐Duration :

☐Site :

☐Character :

☐Radiation :

2. Fever : Yes / No

☐Duration :

☐Type :

☐Associated with chills and rigors :

3. Diarrhoea / Dysentery :

☐Duration :

☐Mucous diarrhea :

☐Blood in stools :

4. Vomiting :

☐Duration / Frequency :

☐Color :

5. Jaundice : Yes / No

6. Cough : Yes / No

7. Distension of abdomen: Yes / No

8. Altered Sensorium : Yes / No

9. Any Other :

PAST HISTORY

Diarrhea : Yes / No
Jaundice : Yes / No
Diabetes : Yes / No

Tuberculosis : Yes / No

Surgery : Yes / No
Trauma : Yes / No

FAMILY HISTORY :
Similar illness :
Other :

PERSONAL HISTORY :

ALCOHOL CONSUMPTION:
Duration
Amount
GENERAL PHYSICAL EXAMINATION :
Pallor : Icterus: Cyanosis:

Clubbing: Pedal edema: Lymphadenopathy:
VITALS:
Temp: Pulse rate:
Blood pressure: Respiratory rate:

LOCAL EXAMINATION :

ABDOMEN:

INSPECTION :

1. Shape : Scaphoid / flat / distended / obese
2. Umbilicus:
3. Swelling:
4. All quadrants moves equally with respiration
5. Dilated viens:yes/no
6. VGP/VIP:
7. hernial orifices:
8. external genitalia:

PALPATION:

1. Tenderness :Present / Absent
2. Rigidity : Present / Absent
3. Liver :Palpable / non palpable
4. Upper limit of percussion inMCL :
5. Lower limit of percussion below costal margin: cms
6. Liver span : cms
7. Borders :sharp / rounded
8. Surface : Smooth / Nodular

RESPIRATORY SYSTEM :

1. Shape and expansion of chest :
2. Air entry :
3. Breath sounds :
4. pleural effusion : Present / Absent

INVESTIGATIONS:

1. Blood :

Hb : gm%

T.C. : c/cmm

D.C. : N %, L %, E %, M %, B %

ESR : mm/hr

Prothrombin time : Sec

INR :

FBS : mg%

Blood urea : mg %

SPECIAL INVESTIGATIONS:

Liver Function Tests:
1. Serum Bilirubin : mg%
2. Albumin : gm%
3. Alkaline phosphatase : IU/L
4. S.G.O.T. IU/L
5. S.G.P.T. IU/L
6. PT : IU/L

RADIOLOGICAL INVESTIGATIONS:
1. Plain X-ray Chest PA view :
2. ULTRASONOGRAPHY ABDOMEN **:**
3.CECT ABDOMEN

TREATMENT :
MEDICAL TREATMENT:

SURGICAL TREATMENT:

COMPLICATIONS :
Early :

Late post operative :

சுய ஒப்புதல் கடிதம்

ஆய்வு செய்யப்படும் தலைப்பு	:	கல்லீரல் சீழ் கட்டி (Clinical Study of Liver abscess)
ஆய்வு செய்யப்படும் துறை	:	பொது அறுவை சிகிச்சைத் துறை
மருத்துவமனை	:	அரசு இராயப்பேட்டை மருத்துவமனை, கீழ்ப்பாக்கம் அரசு மருத்துவக் கல்லூரி, சென்னை
பங்கு பெறுபவரின் பெயர்	:	
பங்கு பெறுபவரின் வயது	:	
பங்கு பெறுபவரின் மருத்துவமனை	:	
எண்	:	

பங்கு பெறுபவர் இதனை ( ) குறிக்கவும்.

1. இந்த மருத்துவ ஆய்வின் விவரங்கள் எனக்கு தெளிவாக விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும் அதற்கான விளக்கங்களை பெறவும் வாய்ப்பு அளிக்கப்படும் என அறிந்து கொண்டேன்.
2. நான் இந்த ஆய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ நான் இந்த ஆய்வில் இருந்து விலக ஆசைப்பட்டால் எந்த தடங்கலும் இன்றி விலகலாம் என்றும் அறிந்து கொண்டேன்.
3. இந்த ஆய்வு சம்மந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் பொழுதோ இந்த ஆய்வில் பங்கு பெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்தேன்.
4. இந்த ஆய்வில் பங்கு கொள்ள நான் சுய நினைவோடும் முழு சம்மதத்தோடும் ஒப்புதல் அளிக்கிறேன்.

பங்கு பெறுபவரின் பெயர் :

பங்கு பெறுபவரின்
கையொப்பம் :

ஆய்வாளரின் பெயர் :

ஆய்வாளரின்
கையொப்பம் :

தேதி :

இடம் :

KEY TO MASTER CHART

-	-	Absent
+	-	Present
ALP	-	Alkaline Phosphatase
B	-	Both lobes
Hb	-	Hemoglobin
L	-	Left lobe
M	-	Multiple abscess
NAD	-	No abnormal defects
P	-	Peritoneal rupture
PT	-	Prothrombin time
RBS	-	Random blood sugar
Pl.eff	-	Pleural Effusion
R	-	Right lobe
S	-	Single
S.bilirubin	-	Serum bilirubin
S. Alb	-	Serum Albumin
WBC	-	White blood count

										1	MASTER CHART													
S.No	Name	I.P NO	Age	sex	fever	abdomin	jaundice	alcoholis	hepatom	Hb(gm/d	WBC	RBS	Urea	.bilirubi	ALP Rai	S.Alb(<3	PT(>20s	Lobe In	Single/M	Treatment	Complic	Recurre	Pus C&S	duration stay
1	Murugan	48970	40	M	+	+	-	Yes	No	10.2	14000	90	39	0.3	No	No	No	R	S	Pigtail drainage	Nil	No	No grwth	15
2	Sarathkumar	48985	28	M	+	-	+	No	Yes	10.6	14500	254	25	4.2	No	No	No	R	S	Pigtail drainage	Nil	Yes	No grwth	5
3	ayubkhan	50524	27	M	+	+	+	Yes	No	12	13600	140	19	2.8	No	No	No	R	S	Aspiration	Nil	No	No grwth	17
4	Nithya	51899	46	F	+	+	-	Yes	No	11.5	13900	120	15	0.7	No	Yes	No	R	S	Pigtail drainage	Nil	No	No grwth	8
5	Maruthupandi	53661	52	M	+	+	+	Yes	Yes	10.6	12900	110	30	4.6	No	No	Yes	B	S	Conservative	Nil	No	No grwth	21
6	valli	55181	43	F	+	-	+	No	No	10.4	15500	324	20	3.2	No	No	No	R	M	Aspiration	Nil	No	Gm-ve	4
7	Munuswamy	56185	64	M	-	+	+	Yes	No	11	9000	124	29	4.2	No	No	No	R	S	Aspiration	Nil	No	No grwth	12
8	Perumal	56441	55	M	+	-	+	Yes	Yes	11.8	13000	140	35	7.4	Yes	No	Yes	R	S	Pigtail drainage	Nil	No	No grwth	9
9	Govindhan	50382	45	M	+	+	-	Yes	No	12.2	14000	80	43	0.8	No	No	No	R	S	Pigtail drainage	Nil	No	No grwth	13
10	Senthil	50618	38	M	+	+	-	Yes	No	10.4	13700	68	43	0.4	No	No	No	R	S	Laparotomy	P	No	No grwth	15
11	Ayyasamy	59934	56	M	+	-	+	Yes	No	10.9	8600	89	49	2.8	Yes	No	No	R	S	Pigtail drainage	Nil	No	No grwth	26
12	velu	61590	43	M	+	+	+	Yes	Yes	10.4	13100	324	39	3.3	No	Yes	No	R	S	Conservative	Nil	No	No grwth	6
13	Rajendiran	65751	65	M	-	+	-	No	No	7.8	12000	89	29	0.3	Yes	No	No	R	M	Conservative	Nil	No	No grwth	15
14	Ram	50626	27	M	+	+	+	Yes	Yes	10.4	12300	120	20	2.5	No	No	No	R	S	Aspiration	Nil	No	No grwth	19
15	Silambarasan	48790	28	M	+	+	+	Yes	Yes	10.5	15000	114	40	2.9	Yes	No	No	R	S	Conservative	Nil	No	No grwth	8
16	Senthilkumar	49780	47	M	+	+	-	Yes	No	11	7000	108	22	0.6	No	No	No	R	S	Pigtail drainage	Nil	Yes	No grwth	18
17	Kaber	49990	30	M	+	-	+	Yes	No	11.2	18000	78	21	2.8	Yes	Yes	No	L	M	laparoscopy	Nil	No	No grwth	16
18	kumar	51220	60	M	+	+	-	Yes	Yes	11.5	10000	77	90	0.7	Yes	No	No	B	S	Conservative	Nil	No	No grwth	12
19	Manikandan	40135	45	M	+	-	+	Yes	No	12.4	15200	209	30	3.6	No	No	Yes	R	S	Conservative	Nil	Yes	No grwth	6
20	Jayakumar	69890	53	M	+	+	-	Yes	No	11.7	11000	89	36	0.7	No	Yes	No	R	S	Aspiration	Nil	No	No grwth	9
21	Mahendiran	50596	38	M	+	+	+	Yes	Yes	10.5	12000	97	38	3.1	Yes	No	No	R	S	Aspiration	Nil	No	No grwth	10
22	Kavi	57541	53	F	+	+	-	No	No	7.8	12500	250	39	0.7	No	No	No	R	S	Conservative	Nil	No	No grwth	12
23	Chinnaraj	66696	42	M	-	+	+	Yes	No	11.3	12400	115	18	2.3	Yes	No	No	R	M	laparotomy	P	No	Staph Aureus	42
24	veeramani	52723	24	M	+	-	+	Yes	Yes	11.4	10050	140	34	3.1	No	No	No	B	S	Conservative	Nil	No	No grwth	17
25	Faisudeen	47576	45	M	-	+	+	Yes	No	10.2	14000	280	14	3.2	No	No	No	R	S	Aspiration	Nil	No	No grwth	8
26	Thangamani	46761	33	M	+	+	+	No	No	10.5	15000	224	19	2.6	No	No	No	R	S	Conservative	Nil	No	No grwth	4
27	kamaraj	46964	40	M	-	+	+	Yes	Yes	12.8	11000	110	33	2.9	Yes	No	No	R	S	Conservative	Nil	No	No grwth	4
28	balaji	47205	55	M	+	+	+	Yes	No	11.3	12500	105	39	3	No	No	No	B	S	Aspiration	Nil	No	No grwth	8
29	ayyammal	46280	45	F	+	+	-	No	Yes	10.5	10500	144	68	0.7	No	No	No	R	S	Aspiration	Nil	No	No grwth	17
30	prabhakar	45282	47	M	+	-	-	Yes	No	10.2	11000	117	20	0.4	Yes	No	No	R	S	laparoscopy	Nil	Yes	No grwth	6
31	selvam	44259	35	M	-	+	-	No	No	10.8	15500	105	69	0.7	No	No	No	R	S	Aspiration	Nil	No	No grwth	13
32	narayanan	44965	58	M	+	+	-	Yes	Yes	11.6	12000	98	59	0.4	Yes	No	No	R	S	Aspiration	Nil	No	No grwth	3
33	harishankar	45331	45	M	+	-	-	Yes	No	11.9	13000	154	26	0.8	No	No	No	R	S	Aspiration	Nil	No	No grwth	6
34	jayaseelan	45578	46	M	+	+	+	Yes	No	13.4	12,500	254	16	3.2	No	No	No	L	M	laparoscopy	Nil	No	Staph	3
35	jayalakshmi	43946	35	F	-	+	-	No	Yes	13.5	13,000	70	28	0.7	No	No	No	R	S	laparotomy	P	No	No grwth	13
36	Arivalagan	43152	25	M	+	-	-	Yes	No	10.2	13,500	90	15	0.8	No	No	No	R	S	Pigtail drainage	Nil	No	No grwth	10
37	mohan	42914	66	M	-	+	-	Yes	No	10	13,000	98	70	0.5	No	No	No	L	S	Aspiration	Nil	No	No grwth	4
38	nandhagopal	46248	38	M	+	-	+	No	Yes	10.4	12,500	106	29	2.6	Yes	No	Yes	R	S	Conservative	Nil	Yes	No grwth	3
39	krishna	49891	51	M	-	+	-	Yes	No	9.6	8,000	227	26	0.4	No	No	No	R	M	laparoscopy	Pl. Eff.	No	No grwth	3
40	kannan	57926	48	M	+	+	-	Yes	Yes	11	22,000	105	130	0.3	No	No	No	R	S	Aspiration	Nil	No	No grwth	7
41	raji	49805	37	M	+	-	+	No	No	11.2	10,500	140	69	3.1	Yes	No	No	B	M	Conservative	Nil	No	No grwth	14
42	radha	58913	60	F	+	+	-	No	No	10.3	11,000	120	19	0.5	No	No	No	L	M	Conservative	Nil	No	No grwth	6
43	karthik	48905	60	M	-	+	-	Yes	Yes	11	13,500	110	29	0.7	No	No	No	R	S	laparotomy	P	No	No grwth	20
44	arul	48783	21	M	+	+	-	Yes	No	10.3	13,000	89	19	0.7	No	No	No	R	S	Pigtail drainage	Nil	No	No grwth	22
45	rajapandi	48862	60	M	-	+	-	No	No	9	12,800	270	29	0.5	Yes	No	No	B	S	Conservative	Nil	No	No grwth	26
46	ramesh	56864	28	M	+	+	-	Yes	Yes	10.8	12,400	85	74	0.5	No	No	No	R	S	Pigtail drainage	Nil	No	No grwth	3
47	deepak	57980	34	M	-	+	-	Yes	No	11.3	13,000	96	10	0.2	No	No	No	L	S	Aspiration	Nil	Yes	No grwth	4
48	gowtham	57993	64	M	-	+	-	No	No	10.5	13,500	86	14	0.8	Yes	No	No	R	M	Conservative	Nil	No	Gm-ve	23
49	raji	58433	45	F	+	+	-	No	No	11.6	9,000	89	30	O.6	No	No	No	R	S	laparoscopy	Nil	No	No grwth	4
50	bharathi	58815	60	F	+	+	+	No	Yes	11.2	10,500	110	33	O.7	No	No	No	R	S	Aspiration	Nil	No	No grwth	13
51	asokan	51175	54	M	+	-	-	No	No	10.4	12,600	120	20	O.6	No	No	No	R	M	Conservative	Nil	No	No grwth	15
52	kalairaj	58862	57	M	-	+	+	No	No	10.6	12,800	70	29	8	Yes	No	No	R	S	Aspiration	Nil	No	No grwth	8
53	baskar	59964	49	M	+	+	-	Yes	Yes	12	13,600	98	15	O.3	No	No	No	R	S	Aspiration	Nil	No	Gm-ve	13
54	murugan	50866	36	M	+	-	+	No	No	11.3	7,000	220	18	2.8	No	No	No	B	M	Conservative	Nil	No	No grwth	9
55	venugopal	50666	56	M	+	-	-	Yes	Yes	10.6	13,500	79	16	O.9	No	No	No	R	S	Aspiration	Nil	No	No grwth	19
56	selvam	57286	48	M	-	+	-	Yes	No	13	14,000	280	29	0.6	Yes	No	No	R	S	Aspiration	Nil	No	No grwth	13
57	perumal	50325	65	M	+	+	-	No	No	9.6	12,800	86	19	O.3	No	No	No	R	S	Conservative	Nil	No	No grwth	5
58	Elumalai	50284	48	M	+	-	+	No	No	12.2	11,000	160	29	3.5	No	No	No	L	S	Aspiration	Pl. Eff.	No	No grwth	22
59	janani	59677	27	F	+	+	+	Yes	No	8.6	13700	80	55	2.8	No	No	No	R	S	laparotomy	P	No	No grwth	32
60	ravi	58129	36	M	+	+	-	No	No	11.2	13,500	78	29	O.4	Yes	No	No	R	S	Conservative	Nil	Yes	No grwth	12